A Dissertation on

STUDY ON ADOLESCENT GYNAECOLOGICAL PROBLEMS

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERSITY CHENNAI.

in partial fulfilment of the regulations for the Award of the degree of

M.S., (Obstetrics & Gynaecology) Branch – II



INSTITUTE OF SOCIAL OBSTETRICS AND GOVT. KASTURBA GANDHI HOSPITAL FOR WOMEN AND CHILDREN

MADRAS MEDICAL COLLEGE
CHENNAI.

APRIL 2015

BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of Dr. RADHE

AKANG on "STUDY ON ADOLESCENT GYNAECOLOGICAL

PROBLEMS" during her M.S., (Obstetrics & Gynaecology) course from

April 2012 to April 2015 at the Institute of Social Obstetrics, Government

Kasturba Gandhi Hospital for Women and Children, Madras Medical

College, Chennai.

Prof.Dr.T.G. Revathy, M.D, D.G.O.,

Professor & Chief of the Department Dept. of Obstetrics & Gynaecology, ISO-KGH

Madras Medical College

Chennai.

Prof.Dr. Baby Vasumathi, M.D., D.G.O.,

Director ISO-KGH

Madras Medical College,

Chennai.

Prof. Dr. R. VIMALA, M.D,

DEAN,

Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai.

Place:

Date:

DECLARATION

I solemnly declare that the dissertation titled "STUDY ON

ADOLESCENT GYNAECOLOGICAL PROBLEMS" is done by me

SOCIAL OBSTETRICS AND INSTITUTE OF GOVT. at

GANDHI HOSPITAL **FOR KASTURBA** WOMEN **AND**

CHILDREN, Madras Medical College under the guidance and

supervision of Prof. Dr.T.G.Revathi M.D., D.G.O., Professor of

Obstetrics and Gynaecology, Madras Medical College, Chennai.

This dissertation is submitted to the Tamil nadu Dr. M.G.R

Medical University towards the partial fulfillment of requirements for the

award of M.S. Degree (Branch II) in Obstetrics and Gynaecology.

Place: Chennai

Date:

Dr. Radhe Akang

ACKNOWLEDGEMENT

I Thank **Dr. R. Vimala, M.D.,** Dean Madras Medical College for permitting me to conduct this study in Institute of Social Obstetrics and Government Kasturba Gandhi Hospital for Women and Children, Chennai.

I owe my sincere thanks to **Prof. Dr.Baby Vasumathi, M.D. D.G.O.** Director, ISO – KGH for her valuable guidance, during the study.

I express my profound gratitude to my guide **Prof.Dr.T.G. Revathy, M.D., D.G.O.,** Professor of Obstetrics and Gynecology for her unwavering support and encouragement.

I also my sincere thanks to **Prof. Dr. S. Dilshath, M.D, D.G.O.,**Former Director, ISO – KGH for her support and encouragement.

My gratitude to my Assistant Professors, Statistician Mr.Ravanan my colleagues and Hospital Staff and patients for enabling me to complete the study.

CONTENTS

Sl.No.	TOPICS	Page No.
1.	INTRODUCTION	1
2.	AIM AND OBJECTIVES OF THE STUDY	4
3.	REVIEW OF LITERATURE	5
4.	MATERIALS AND METHODS	51
6.	OBSERVATIONS AND RESULTS	54
7.	DISCUSSION	96
8.	SUMMARY	102
9.	CONCLUSION	104
	BIBLIOGRAPHY	
	ANNEXURE	
	PROFORMA	
	ABBREVIATION	
	INFORMATION SHEET	
	CONSENT FORM	
	ETHICAL COMMITTEE APPROVAL FORM	
	TURN IT IN SCREEN SHOT	
	MASTER CHART	

INTRODUCTION

Puberty coined from latin word 'Pubertus' meaning 'grown up' (Foster 1992). "Puberty is the period of physiological changes in secondary sexual characteristics development and their attainment of capability of sexual reproduction". Puberty not only deals with mere physiological changes but there is also tremendous psychological and emotional changes in the life of the adolescent. Adolescence, in simple words is nothing but the phase of life during which a carefree child becomes the responsible adult.

"WHO defines adolescence as the period in human growth and development that occurs after childhood and before adulthood, from age 10-19 years". It is an ongoing process i.e; the psychological and physiological changes which occur may begin before and even can continue after this age(**Progress in Obstetrics and Gynaecology, vol 12; John Studd**).

The biological determinant of adolescence are universal, however there may be variation in the duration, defining charactecristics of this phase across time, demographic area, culture and socioeconomic conditions. Over the passage of time, there have been many changes in the pattern of this period like the time of onset of puberty, urbanization, changing attitude towards sexuality, behavior etc.

Adolescence in girls has been regonised as a special phase in their life. It requires a specific approach and special attention. This period of transition makes them vulnerable to various problems e.g., general, reproductive health related, sexually related and psychological problems. This period in a girl's life is the preparation for safe motherhood. These adolescent girls are the direct reproducer for the future generation. So, the health of these girls not only influences her own health but, also the health of future generation.

Reproductive health related problems of adolescent girls has its own special space in the spectrum of gynaecological problems of all ages. This is because of its unique presentation and association with emotional and psychological factors.

Today, adolescent forms 20.9% of total Indian population (Population Census of India 2011) and their health determines the health of the nation. Adolescent sexual and reproductive health forms a major component of global burden of sexual ill health but has been historically overlooked in terms of sexual and reproductive health intervention. Though, FOGSI has dedicated the year 1999 as the year for

adolescent health and addressed the need for adolescent health clinics but still it remains a sub specialised area of gynaecology which still needs a great deal of attention.

With this preview, my thesis titled 'Study on adolescent gynaecological problems', I have made an attempt to review and analyse various gynaecological problems and factors responsible for development and progression of these problems in adolescent girls attending gyanecological OPD and emergency casualty in ISO & KGH Hospital

AIM OF THE STUDY

To study various gynaecological problems encountered in adolescent girls who attended the gynaecological out patient department of Obstetric and Gynaecology in ISO & Kasturba Gandhi hospital for women and children, Triplicane.

To analyse different causes of the gynaecological problems in adolescent girls in age 13-19 years.

REVIEW OF LITERATURE

Puberty is the phase of life in which there is a transition from childhood to adulthood. There is development of secondary sexual characteristics, onset of menstruation and also the sexual reproduction capability. Puberty includes a series of events which are predictable but vary in timings, pace and also in sequence. It initiates through a cascade of endocrinal changes that leads to sexual maturation and reproductive capability (Patton and Viner 2007)

TIMING OF PUBERTY:

The age of puberty occurs earlier these days than in the past. It generally starts earlier in girls than in boys. There are various determinants of the timing of the puberty onset like nutritional status, geographical location, general health, exposure to light, psychological state. But the major determinant is no doubt genetics. The average age of menarche has declined in recent years, according to a study published in the journal Paediatrics. This decline in the age of menarche has also been attributed to the improved nutrition and healthier living condition (Patton and Viner 2007).

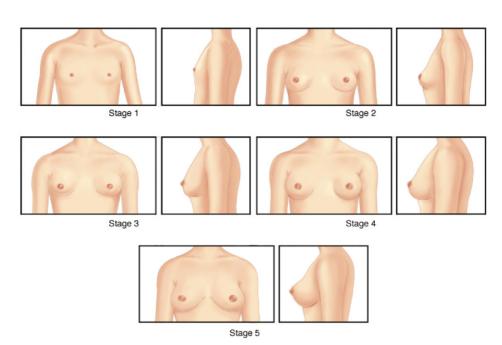
There has been various researches showing influence of ethnic backgrounds, body mass index, family income on the onset of puberty. Menarche occurs earliest in those girls whose BMI falls under the obese category(upto 20% above normal weight for age), followed by normal weight, then underweight and lastly the girls who suffers from anorexia. But, it is to be noted that the pathological obesity is associated with delayed puberty rather than earlier onset. Conditions like chronic illness, strenuous physical activity as in atheletes, malnutrition are responsible for delayed puberty in some. Puberty starts earlier in those staying in low altitude areas than those in high altitude.

PHYSICAL CHANGES OF PUBERTY:

The most frequently staging system to describe the physical changes of puberty was described first by **Marshall and Tanner in the year 1969**. The physical change constitute three main components:

- a) Secondary sexual characteristics
- b) Gonadal development and function leading to menarche
- c) Growth spurt

The typical sequence includes the larche, adrenarche, growth spurt and menarche



- Stage 1 Preadolescent: juvenile breast with elevated papilla and small flat areola.
- Stage 2 The breast bud forms under the influence of hormonal stimulation. The papilla and areola elevate as a small mound, and the areolar diameter increases.

 Stage 3 Continued enlargement of the breast bud further elevates the papilla. The areola continues to enlarge; no separation
- of breast contours is noted.
- Stage 4 The areola and papilla separate from the contour of the breast to form a secondary mound.
- Stage 5 Mature: areolar mound recedes into the general contour of the breast; papilla continues to project.

Source: Kappy MS, Allen DB, Geffner ME: Pediatric Practice: Endocrinology: www.accesspediatrics.com Copyright © The McGraw-Hill Companies, Inc. All rights reserved.

PHASES OF PUBERTAL GROWTH:

Phase 1 (Thelarche):

The first sign of puberty is the development of breast. It indicates the competency of HPO axis. Marshall and Tanner described the breast changes in five stages:-

Tanner stage 1(B_1): Pre pubertal state, no palpable breast tissue, with areolar less than 2 cm in diameter.

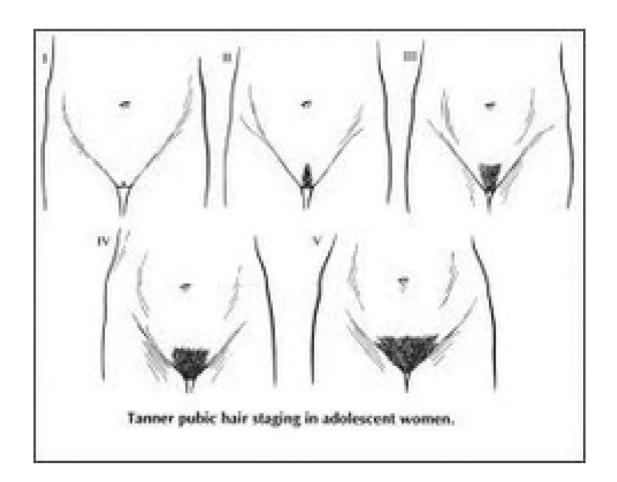
Tanner stage $2(B_2)$: Breast budding occurs, visible and palpable mould of breast tissue

Tanner stage 3(B_3): Further enlargement and elevation of the entire breast.

Tanner stage $4(B_4)$: Projection of areola and papillae to form a secondary mould above the level of breast.

Tanner stage $5(B_5)$: Breast is mature in contour and proportion. Areola receeds into the general contour of the breast.

According to the Copenhagen Puberty study (Paediatrics Vol.123, No. 5, May 2005), there is a decline in the age at breast development.



Phase 2 (Adrenarche):

It indicates the rise of adrenal androgen secretion with a rise in blood level of DHEAS and androstenedione. This ultimately results in the growth of pubic and axillary hair(pubarche).

Tanner stage 1 : No sexually stimulated pubi hair

Tanner stage 2: First appearance of sparse growth of long coarse and crinkly hair along the labia majora.

Tanner stage 3: Hair are coarser, darker and curlier extending into the the mons pubis

Tanner stage 4: Adult type of hair but does not typically extends to inner side of the thighs.

Tanner stage 5 : Adult hair type extending even to inner aspects of the thighs

Phase 3 (Growth spurt):

Girls usually reaches peak height velocity in puberty 2 years earlier as compared to the boys. Growth hormone, insulin like growth factor (IGF-1), and gonadal steroid mainly estrogen plays an major roles. Growth spurts preceeds about one years prior to menarche.

Phase 4 (Menarche):

This is the first menstrual cycle which follows the larche by about 2 years. The initial menstrual cycles are anovulatory, this is due to the immature HPO axis which may take 1 -5 years to completely mature. Onset of menstruation depends on various factors.

Average age of menarche in India

Study	Age of Menarche
Prabhakar et al, 1972	13.2 years
Ghosh et al, 1973	13.2 years
Singh <i>et al</i> , 1987	13.4 years
Grover <i>et al</i> , 1989	12.6 years
Hedge et al , 1990	13.5 years

Phase 5:

This is includes complete sexual maturity with ovulatory menstrual cycle, full breast development and adult pattern of pubic and axillary hair and complete pubertal growth.

PHASE	DESCRIPTION	MEAN AGE(INDIAN STANDARD)
I	Thelarche	10-11 yrs
II	Adrenarche	11-12 yrs
III	Growth spurt /Peak growth velocity	12-13 yrs
IV	Menarche	11-14 yrs
V	Mature sexual hair and breast	14-15 yrs

GONADAL DEVELOPMENT AND FUNCTION

During fourth and fifth month of fetal life, there is appearance of primordial follicles. It constitutes the life long store of follicles for an individual. Thereafter, these follicles grow and enters the antral stage, but before menarche they undergo the process of atresia. During puberty, follicular growth occurs and forms oocytes which enlarges and forms granulosa cells. These follicles undergoes luteinisation during the

reproductive life, forming corpus luteum. It is the main source of gonadal steroids after ovulation. In the absence of fertilization, these follicles undergoes dedifferentiation and cytolysis.

Ultrasonographic studies show that during the pubertal growth, there is an increase in the corpus of uterus form an initial tubular form to bulbous form. There is increase in the length of uterus from 2-3 cms to 5-8 cms. After the onset of puberty, there is increase in ovarian volume from 0.7-0.9 ml to 2-9 ml. According to **Yen SSC in 1980,** " normal ovarian volume is 4 ml in age group of 12-20 years and around 7.5 ml in 20-32 years age".

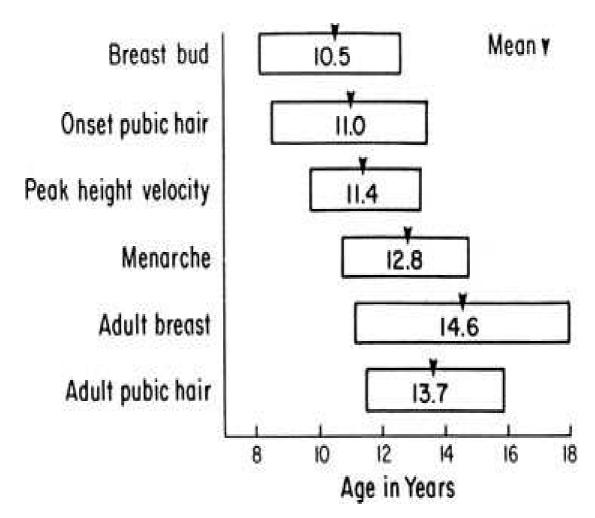
CONTROL OF GROWTH SPURT IN ADOLESCENT

The greatest growth occurs in infancy, thereafter it reduces to minimal till the beginning of puberty. During puberty, a significant proportion about 17-18% gain in the adult height (Leon Speroff & Marca Fritz, 8th edition 2011). Growth in puberty is controlled by complex hormonal mechanism involving growth hormone, insulin like growth factor, gonadal steroids and thyroid hormones.

- a) The most important decisive factor of pubertal growth rate is the rate at which circulating GH level increases. GH acts by secretion of IGF-I ,which promotes growth and differentiation.
- b) Gonadal steroid act via induction of GH secretion and limiting the adult height by stimulating epiphyseal fusion.

Marshall and Tanner (1969) showed that the mean age for attaining peak height velocity in girls was between 12-14 years.

SEQUENCE OF PUBERTAL MILESTONES



BEHAVIOURAL CHANGES OF PUBERTY:

Adolescence is a difficult time for both the child as well as parents as it involves not only profound physical changes but also triggers emotional, cognitive, and behavioural changes **Patton and Viner 2007.** This alternation in the cognitive processing clearly are associated with change in attitude of adolescents to not only themselves but also towards others. The psychosocial changes during puberty predispose to increased incidence of depression, twice more common in girls as in boys.

HORMONAL AND METABOLIC CHANGES IN PUBERTY:

Puberty is the stage which extends from sexual differentiation and the ontogeny of the hypothalamic-pituitary- gonadal axis to completion of the sexual maturation. Development of hypothalamic-pituitary- portal venous axis results in dramatic rise in FSH and LH in fetal pituitary gland, but it falls around term due to negative feedback effects of estrogen and progesterone derived from placenta. It remains suppressed in early infancy. This pre-pubertal pause lasts about approximately one decade. The HPO axis in girls develop in two distinct stages during puberty:

- 1) Gonadal steroid dependent negative feedback mechanism
- 2) Intrinsic CNS inhibitory mechanism.

COMMON PROBLEMS ASSOCIATED WITH PUBERTY

1) Delayed Puberty:

Delayed puberty in girls may be defined as the absence of physical manifestation of puberty by the age of 13 years, or the absence of menstruation by the age of 16 years in the presence normal sexual growth.

Causes includes:-

- a) Constitutional delay (commonest cause)
- b) Hypogonadotropic-Hypogonadism
- c) Hypergonadism-Hypogonadism (Primary gonadal failure)

Constitutional delay which is by far the most common cause of the delayed puberty is considered to be due to physiological immaturity or functional deficiency of gonadotropin hormone. Such girls frequently give history of delayed menarche in their mother or sisters.

Evaluation of Delayed Pubertal Development:

- 1) Evaluation starts with careful history taking, which must also includes complete family history. Emphasis should be given on the age at the pubertal milestones in other siblings and also parents too.
- 2) A thorough physical examination.
- 3) Bone age measurement.

Laboratory evaluation and Imaging:

- 1) Measurement of serum FSH, LH, Estradiol, TSH, free thyroxine(T4), prolactin, DHEA-S(wherever indicated).
- 2) X-ray of bone
- 3) Karyotype
- 4) MRI or Cranial CT scan (when indicated)

Treatment:

- 1) **Reassurance and psychological support** to the girl as well as the parents.
- 2) **Correct specific cause** if diagnosed such as thyroid hormone supplementation in case of hypothyroidism, dopamine agonist in hyperprolactinemia, neurosurgical procedure in operable cranial tumors etc.
- 3) **Hormonal therapy** It promotes the age related secondary sexual development ad also induce growth.

Oral estrogen 0.25- 0.5 mg is given, increasing gradually at an interval of 3-6 month according to response (Tanner stage, bone age).

Progestin in general is started only after the commencement of menses, or after 12-24 months of estrogen therapy in order to ensure that it does not interfere with the development process of breast. After the accomplishment of breast development and establishment of menstruation, hormonal therapy can be discontinued for 1-3 months at intervals, in order to observe commencement of spontaneous menses, as expected in case of constitutional delay of puberty. Persistent hypogonadism even after age of 18 years suggests congenital GnRH deficiency.

2) PRECOCIOUS PUBERTY:

It is defined as the occurrence of pubertal growth ≥ 2.5 standard deviations earlier than the average age. It is more common among girls than in boys (**Speroff** et al 1999).

Classification:-

a) Gonadotropin- dependent precocious puberty- also known as "Central or True Precocious Puberty". It is due to early maturation and activation of HPO axis, and development of both breast and pubic hair. They are isosexual, consistent with the actual gender of the child.

b) Gonadotropin- independent precocious puberty- also known as "Peripheral or Pseudo Precocious Puberty". It is the result of early exposure of sex steroid, independent of GnRH and gonadotropins ending up in early sexual development. They may be isosexual or contrasexual.

Evaluation:

a) Clinical, medical and family history

b) Physical examination including Tanner staging and measurement of bone age.

c) Endocrinal evaluation : Basal gonadotropin level

: Serum LH

: Thyroid function test

: GnRH stimulation test

Serum estradiol, DHEA-S, testosterone, cortisol in gonadotropin- independent precocious puberty.

d) Imaging : i) MRI brain

ii) Abdominal and pelvic ultrasonography.

Treatment:

The aim of treatment is to slow down the development until the girl is of normal pubertal age, to maximize adult height, and to reduce risk of psychological problems associated with early sexual maturation. Reassurance and psychological support to both the adolescent girl and parents is an important aspect of the treatment.

GnRH agonist therapy is the drug of choice in cases of GnRH dependent precocious puberty (Michael, Thomas & Robert et al).

MENSTRUAL CYCLES:

Menarche is coined from greek word 'men' meaning 'moon' and 'arkhe' meaning 'beginning'. The normal menstrual cycle results from complex feedback mechanism involving the hypothalamus, pituitary, ovary and uterus. According to Zarcharias et al (1970), regular menstruation was shown to occur little more than a year after menarche. Most of the early menstrual cycles are anovulatory. In Vollman's series (1977), 55% of cycles were anovulatory at gynaecological age 1-2, decreasing to 3% at gynaecological age 11-12. Anovulatory cycles usually persists for 12-18 months but can even prolong for 4-5 yrs after the menarche. There are variation in the interval between periods, their duration and the amount of the blood loss in most of the girls. Such

variation are within the physiological limits and requires basic investigation and proper counseling.

According to **Metcalfal 1988**, "menstrual dysfunction is common in the first 18 months following menarche. As many as 58% of adolescents with gynaecological complaints will present as menstrual disorder (**Dramusic** *et al* 1991).

MENSTRUAL DISORDERS IN ADOLESCENTS:

Types of menstrual disorders

- 1. Amenorrhoea
- 2. Dysmenorrhoea
- 3. Irregular menstrual cycles
 - a) Oligomenorrhoea
 - b) Polymenorrhoea
- 4. Menorrhagia

AMENORRHOEA IN ADOLESCENTS:

Amenorrhoea meaning absence of menstruation. It is a symptom, not a disease, having various causes. Any of the following criteria needs to be fulfilled to be evaluated for amenorrhoea as per **Fertility & Sterility, 2006**

- a) Absence of menses by age of 13 years in the absence of development of secondary sexual character.
- b) Absence of menses by age of 15 years irrespective of presence of secondary sexual character.
- c) Absence of menses in a previously menstruated women, for an interval of time equivalent to a total of at least three previously, or 6 month.

First two criteria falls under primary amenorrhoea, while the third one under secondary amenorrhoea.

Etiologies of Amenorrhoea:

Primary amenorrhoea is usually the result of genetic or anatomic abnormality. Etiologies are:-

- i) Abnormalities of HPO axis
 - Constitutional delay
 - Functional (stress, exercise, anorexia etc)
 - Kallmann's syndrome
 - Gonadal dysgenesis
 - Primary ovarian failure

- ii) Chromosomal abnormalities
- iii) Anatomic abnormalities- e.g. Imperforate hymen, absent vagina, transverse vaginal septum, MRKH Syndrome etc.
- iv) Medical disorders Chronic illness : Juvenile diabetes

: Tuberculosis

: Thyroid disorders

- v) Enzyme deficiency 5α reductase deficiency
 - 17,20 desmolase deficiency

Secondary amenorrhoea are more commoner than primary amenorrhoea. Causes are divided into:-

- i) Hypothalamic dysfunction Stress, excessive weight gain / loss, tumors.
- ii) Pituitary disorders Cushing's disease, adenomas.
- iii) Ovarian disorders PCOS, Premature ovarian failure.
- iv) Uterine diseases Tubercular endometritis
- v) Medical disorders Thyroid dysfunction, chronic illness
- vi) Others Pregnancy, lactation.

Approach to Patient of Amenorrhoea

- 1. **History taking:** A thorough history regarding the patient as well as family history should be elicited. It must include girl's menstrual history, physical growth history, previous medical and surgical history, etc.
- 2. **Physical examination:** Detail examination of patient including the height / weight / BMI / Nutrition / anthropometry and growth charts, thyroid, breast (Tanner staging), hair distribution, secondary sexual character ,etc.

3. Pelvic examination:

- a) Examination of external genitalia: Tanner staging of pubic hair/virilization / ambiguous genitalia.
- b) Examination of internal genitalia: Presence or absence of uterus /vaginal septum/ imperforate hymen/absent vagina.
- 4) **Pregnancy** to be ruled out
- 5) **Tuberculosis** should be looked for.
- 6) Hormonal evaluation:
 - a) FSH/LH
 - b) Serum Prolactin

- c) Plasma testosterone
- d) DHEAS
- e) TSH, T₃ and T₄
- f) Serum Progesterone
- g) Estrogen

The hormonal tests must be done at an appropriate time of menstrual cycle.

7) **USG**: It is an important diagnostic tool.

Hormonal assessment:

- Serum FSH, LH, estradiol if low then the cause lies in the pituitary.
- If FSH and LH are high, estrogen is low then
 - a) Evaluate for the bone age, if lower than chronological age, then wait and watch.
 - b) Kryotyping to rule out Turner's syndrome.

Treatment:

The main objective of treatment of Primary amenorrhoea is to correct the underlying pathology, if possible, and to prevent complication of the disease process. **ACOG** recommends initial reproductive health visit at 13-15 years of age.

Principles in the management of Primary amenorrhoea:

1. Identification of underlying disease and treating the cause.

2. Reassurance:

Adolescent girls and her parents should be reassured regarding anticipated normal pubertal development in case of delay in both growth and puberty.

3. Short –term therapy with sex steroids (estrogens)

This may be needed in girls with Combination delay of growth and puberty (CDGP) to trigger the onset of puberty.

4. Induction and maintenance of puberty:

To prevent the early epiphyseal clsure and also avoid psychological embarrassment due to the peer pressure, the therapy should be started at 13 years of age.

The starting dose of ethinyl estradiol is 1-2 mg mg/day, gradually increased every 2-3 months. Progestin should be added to either on full breast development or after the break through bleeding occurs.

5) Growth promoting and potentiating strategies:

In cases like Turner's syndrome (TS) and Prader-Willi syndrome (PWS) with abnormal physical growth like short stature is managed with growth hormone therapy. And to assure maximum gain in height, estrogen replacement therapy can be timely added for optimum synergistic action and preventing early epiphyseal closure.

6) Prevention of osteoporosis:

Adolescence is a critical period of bone acquisition depending on nutrition, lifestyle and estrogen. Girls with permanent gonadal failure need estrogen for not only for maintenance of puberty but also for prevention of osteoporosis.

7) Fertility:

With newer advancement in the reproductive medicine, a large number of patients with pubertal disorders have been assured of future fertility. Some of the ways are :-

- Induction of ovulation with gonadotropins in hypogonadotropic hypogonadism
- ART with donor eggs in hypergonadotropic hypogonadism
- Trans-sphenoidal surgery in Pituitary adenoma
- Bromocriptine Hyperprolactinemia
- Mullerian agenesis IVF with surrogacy using woman's own egg and husband's sperm.

DYSMENORRHOEA:

Dysmenorrhoea is a gynaecological medical condition which is characterized by severe uterine pain during menses. In around 5-10%. It is severe enough to unable them to attend their normal day today work, attend school or college (Sheil & Turner 1996). This kind of dysmenorrhoea may lead to fear psychosis in adolescents (Dickens 1974).

Study by Sheil & Turner, 1996 says 20% of these adolescents have a family history of dysmenorrhoea either in their mother or sister.

Dysmenorrhoea can be mild, moderate, or severe depending upon the severity, or can be of primary or secondary type depending upon the cause. General measures like counseling and reassurance, lifestyle modification are the simple measures to overcome this problem. Complete history taking is the most important aspect in the management of such cases, includes details about the menses and associated problems. But depending upon the severity and associated underlying cause if needed, to be treated by pharmacological or surgical management.

Diffentiating features between Primary and Secondary dysmenorrhoea

Features	Primary	Secondary
Onset	First day of menses	Prior to to menses
Duration	12-24 hours	Throughout the menses
Ovulation	Present	Absent
Pathology	Usually absent	Uterine/ pelvic pathology present
Aetiology	Increased PGE_2 & $PGF_{2\alpha}$	Congestion
Prevalence	Most common	Less common
Severity	In 5-10%	Not so severe

IRREGULAR MENSTRUAL CYCLE

Menstrual irregularities are the commonest gynaecological complain in adolescents (**Dramusic** *et al* **1991**). In about 95% of

abnormal vaginal bleeding, the cause is dysfunctional uterine bleeding. DUB is a subset of AUB which is defined as excessive, prolonged, or unpatterned bleeding from the uterine endometrium without an organic cause. These complains varies from excessive bleeding that is regular (menorrhagia), irregular (metrorhagia), combination both (menometrorrhagia) intermittent cyclical bleeding or sparse (oligomenorrhoea). The treatment of these disorders varies from mere observation to pharmacological and/ or surgical management. About 45% of adolescent girls have an anovulatory cycles for approximately 1-2 years after the onset of menarche (Sheil & Turner 1996), which leads to irregular menstrual cycles. There are mainly two types of irregular cycles:

- a) Oligomenorrhoea
- b) Polymenorrhoea

OLIGOMENORRHOEA:

Oligomenorrhoea is infrequent menstruation. It is defined as the periods occurring at a interval of more than 35 days for atleast 6 months in a previous normal cycle. This in adolescents are commonly due to anovulation but if the history and clinical examination is suggestive of any organic cause further evaluation may be required.

Causes of Oligomenorrhoea:

- a) Emotional stress and exercise
- b) PCOS
- c) Thyroid dysfunction
- d) Anorexia nervosa
- e) Adrenal hyperplasia
- f) Pituitary tumors
- g) Excessive weight loss
- h) Obesity

Treatment:

1) Reassurance:

Adolescents and also their parents needs to be counseled properly regarding the problem and reassured that this is usually self limiting and this is not going to affect their fertility in future, if no pathological cause is observed.

2) Hypothalamic or Pituitary causes:

Due to the rise of expectation from parents, society and peer pressure these adolescents are in constant physical and psychological stresses leading to defect in pulsatile secretion of GnRH, which inturn ends up in oligomenorrhoea. So, this can be

managed with proper counseling and behavioral therapy. No drugs are required for regularization of menstruation.

3) Hyperprolactinaemia:

Hyperprolactinaemia in adolescents is rare but if present can be treated with medical drugs like cabergoline, bromocriptine and definitive surgery for prolactinomas, if diagnosed.

POLYCYSTIC OVARIAN SYNDROME (PCOS)

Hyperandrogenism is a frequent cause of oligomenorrhoea in adolescence. The most common form is the syndrome of PCOS. PCOS is a heterogenous disease. It requires special mentioning because they are common in adolescent population.

Several studies have been performed regarding pathophysiology of PCOS but till date no final consensus have been agreed although recently familial clusters of PCOS is reported- suggesting a major component of its etiology. **Steven Frank study** suggests the involvement of two key genes in the etiology of PCOS, the steroid synthesis gene CYPIIa and insulin gene- Variable number tandem repeats (VNTR).

PCOS comprise of a wide spectrum of signs which ranges from presence of multicystic ovarian morphology detected by USG, to obesity,

menstrual irregularities, infertility and to even cardiovascular disorders – all due to metabolic disturbances involving increased level of LH, insulin and androgen and dyslipidemia.

Pre-pubertal ovaries are usually multicystic. These multicystic ovaries are normal findings in the evolution of mature hypothalamic-pituitary-gonadol axis, as described by **Merrill.**

Therefore, PCOS is defined as a dysfunctional condition of the ovary in which there is increased LH dependent secretion of androgen from hyperplastic theca and stromal cells of ovary.

Clinical features of PCOS:-

- a) Hirsutism
- b) Menstrual irregularities
- c) Central obesity
- d) Infertility
- e) Pelvic examination(if done) often normal, as it is uncommon to palpate enlarged ovaries in teenagers.

Rotterdam criteria (2003) suggests presence of at least two criteria to be diagnosed as PCOS, these criteria are :-

- i) Oligo/ amenorrhoea, anovulation, infertility
- ii) Hirsuitism
- iii) USG features of PCOS as described above.

Laboratory Findings:

Endocrinal Screening:

Prolactin and TSH levels are tested to rule out pituitary or thyroid disease as an etiology of anovulation.

In PCOS there is -

- Normal or increased LH
- Normal or increased 17 ketosteroid
- Normal or increased FSH
- Normal or increased E2
- Increased LH/FSH ratio

Sonographic Findings:

Typical USG findings in PCOS

- i) No. of cyst = >10mm pushed periphery (Necklace pattern)
- ii) Abnormal stroma
- iii) Uterine width: ovarian length=<1,
- iv) Increased ovarian volume(> 10 ml),
- v) Increased ovarian roundness index=>0.7

Ovarian Histology:

Multiple microcysts with atretic and immature follicles, fibrosis thickening, and sclerosis of ovarian capsule.

Treatment: Depends on various factors such as:

- Age of girl
- Presence or absence of acne, hirsutism, obesity
- Menstrual irregularities
- Presence or absence of hyperinsulinemia or hyperandrogenism or high LH
- Prevention of long-term sequelae.

The purpose of treatment is to-

- Treat menstrual irregularities
- Treat hirsutism
- Treat infertility
- To prevent long term consequences of PCOS.

Thus, the treatment is modified or prescribed as per the requirement of women. It is subdivided into:-

- a) Life style modification
- b) Medical management
- c) Surgical management

Lifestyle modification:

It include weight reduction, exercise, low calorie diet intake, abandoning cigarette smoking etc. Weight reduction has beneficial effects on menstrual abnormalities as well as in hirsutism (Guzick, Wing, Berga et al 1989). Studies have shown reduction in the level of (a) Serum free testosterone (b) Insulin and insulin like growth factor I(IGF-I) and (c) increase in the level of SHBG due to intake of very low energy diet in girls with PCOS. It has been shown that weight reduction decreased

ovarian P450c17a activity and cause decrease serum free testosterone level in obese girls with PCOS.

Weight loss causes for decrease in both hyperinsulinemia and insulin resistance, increases SHBG and thus results in overall reduction in hyperandrogenism.

Medical Management:

Most commonly used are metformin which reduces hyperinsulinemia and hyperandrogenism independent of changes in body weight (obesity) and OCPs for regularization of periods. The usual dose of metformin is 500 mg thrice daily and can be gradually increased if required. But girl should be cautioned that it make take 6 months or more for complete results. And, patients should also be cautioned that it may recur, later in reproductive life.

Surgical Management:

Surgical management is reserved only in following cases: -

- Medical therapy fails
- Hyperstimulation following medical treatment
- Infertility
- Previous pregnancy loss

Surgical procedure comprises of:-

- a) Wedge resection of ovary
- b) Laparoscopic ovarian puncture- for not more than four cysts in each ovary either by laser or by unipolar electrocautery.

But these surgical procedures have disadvantages of forming pelvic adhesion postoperatively which may further add on to reduction in fertility rate.

SEQUELAE OF PCOS

Long- term sequelae in PCOS is important to know because mainly two reasons:-

- Higher incidence of PCOS in adolescent (20%) diagnosed by USG
- Because of its impact on the future reproductive life.

Complication of PCOS

- a) Short- term sequelae
 - i) Obesity
 - ii) Hirsutism
 - iii) Menstrual irregularities
 - iv) Infertility

- b) Long- term sequelae
 - i) Diabetes
 - ii) Cardiovascular disease
 - iii) Osteoporosis
 - iv) Cancer

POLYMENORRHOEA

Polymenorrhoea is the least common menstrual complaint in this age group. It is usually caused by luteal phase defect (LPD). Adolescents with such complaints are treated with progesterone support in luteal phase. OCPs are used to regularize the cycles.

PUBERTY MENORRHAGIA

Puberty menorrhagia is the excessive menstrual blood loss between menarche and 19 years of age. Menorrhagia is defined as menstrual loss greater than 80 ml. Anovulation and bleeding disorders make up the vast majority of menorrhagia in adolescent. In adolescents, the HPO axis takes time to mature after menarche, which can lead to anovulation.

In first 2 years after menarche, 55-82% 0f cycles are anovulatory, and by fourth and fifth year this decreases to 20% of cycles being anovulatory. These patients generally lack the positive feedback

mechanism necessary to initiate an LH surge and subsequent ovulation despite increased follicular estrogen levels. But, the negative estrogen mechanism is intact in these patients. These are hypothalamic problems rather than pituitary as indicated by normal GnRH stimulation. Unopposed estrogen stimulates endometrial growth, which ultimately outgrows its blood supply. The endometrium becomes excessively thickened and unstable , and the lining breaks down irregularly and unpredictably. This leads to heavy bleeding and prolonged menstruation.

Causes of Puberty Menorrhagia:

- Anovulatory uterine bleeding
- Coagulation disorders Von willebrands disease (32-100%)
 - Idiopathic thrombocytopenic purpura
 - Platelet disorder-e.g. Bernard –Soulier
 Syndrome (51%), Glanzmann's
 thrombasthenia(98%)
- Endometrial tuberculosis
- Thyroid dysfunction
- Pregnancy (In cases of heavy bleeding, adolescent pregnancy should always be ruled out.)

History & Physical Examination:

The evaluation of the adolescent with menorrhagia should begin with detailed history. It is important to assess family history for presence of any known bleeding disorders. Upon physical examination, height, weight, BMI, orthostatic blood pressure and pulse should be recorded. Examination of major body systems gives insight to endocrinological or systemic disease.

Investigation:

- 1) Complete blood count,
- 2) coagulation profile (bleeding time, clotting time, PT, ApTT),
- 3) Thyroid function test(TSH ,FT4,FT3)
- 4) Ultrasonography of pelvis are useful parameters.

If history indicates a bleeding disorder, if bleeding is severe, prolonged or associated with menarche, or if initial screening is abnormal, specific laboratory test for coagulation defects should be done. In developing countries like of ours, where tuberculosis is still highly prevalent, it is a routine test to rule out tuberculosis by laboratory tests like montoux, chest X-ray, and even TB-PCR of the first day menstrual blood.

Treatment:

The goal of initial assessment is to determine which adolescent needs treatment and which adolescent can be observed until the maturation of HPO axis results in regular normal menses.

- 1) Counseling and reassurance In most of girls this is the only therapy required.
- 2) **Anaemia** is corrected with iron supplementation and high protein diet and if required blood transfusion is suggested.
- 3) **NSAIDS** like mefenamic acids, antifibrinolytic such as tranexamic acid and aminoaproic acid, can reduce menstrual blood loss by 50%.
- 4) **Acute bleeding** is stopped by 5-10 mg of norethisterone medroxyprogesterone orally. Later on, cyclic progesterone therapy is recommended
- 5) **Hormonal therapy**: Hormonal therapy is the most effective therapy, with more than 93% of adolescents responding to some form of hormonal treatment. Indication for start of hormonal therapy are as follows:-

- a) Anaemia
- b) Recurrence
- c) Restricted routine activities

The initial treatment is the use of oral medroxy progesterone acetate followed by the use of combined oral contraceptive pills. OCPs can also be used as a first line of treatment in treating menorrhagia in adolescents with bleeding disorders.

If OCPs are contraindicated, or the patient or family does not wish to start oral contraception, cyclic progestins can be used. Oral medroxyprogesterone acetate 5mg can be given for 10-14 days or 21 days each month to induce a withdrawal bleeding that is cyclic and predictable. This pattern is continued for 3-6 months.

6) **Diagnostic curettage (Sheil & Turner 1996)** is rarely indicated in non responder to hormonal therapy in these age group patients. If endometrial hyperplasia is seen then she is put on progesterone for a period of 6-12 months, after which she re-evaluated.

Careful assessment and prompt recognition is important in the adolescent with menorrhagia. No single therapy or treatment is universal, and it must be tailored to the adolescent and clinical situation.

INFECTION IN ADOLESCENT:

Due to lack of oestrogen and alkaline vaginal secretions, young girls are more prone to infection in the pre menarche period. Vaginal discharge is generally the result of infection caused by non specific causes, resulting from poor hygiene or as a result of specific infection.

Aetiology:

1) Chemical / Irritant (or Allergic)

2) Poor hygiene:

Poor personal hygiene may lead to candidal vulvovaginitis, vulval irritation may follow worm infection like pin worms, thread worms secondary to anorectal contamination.

3) **Infection**:

Infection of vulva and vagina i.e, vulvoginitis most commonly due to the infection caused by the normal vaginal flora, which includes mixed aerobes and anaerobes, commonly staphylococcus epidermidis, lactobacilli, bacteroides etc. Sexually transmitted infections like that caused by Neisseria gonorrhoea, Chlamydia trachomatis, Trichomonas vaginilis.

4) Foreign bodies

5) Sexual abuse

Diagnosis:

- a) Detailed history including sexual ativity, type of discharge, use of antibiotics, concomitant infection elsewhere in body, treatment taken etc.
- b) Clinical examination with utmost care. P/R Examination may be required.
- c) Vaginal or urethral discharge collected and wet smear examined in microscope.
- d) Examination under anaesthesia may be required if suspicion of foreign body in vagina or foul smelling discharge is present.

Management:

- ✓ Appropriate antibiotics for specific organism.
- ✓ Non specific Infections : improve hygiene of private parts. Local application of estrogen cream 0.10% for 2-3 weeks.
- ✓ Specific infections : Trichomoniasis : Metronidazole 20 mg
 three times a day for 7 days Alternative
 single dose of 2 gm of metronidazole.

✓ Candidiasis

: Oral nystatin 5 lac units three times a day for two weeks or oral fluconazole.

Locally clotrimazole or miconazole cream for 6-12 days.

OVARIAN MALIGNANCIES IN ADOLESCENT:

Total incidence of ovarian cancer in adolescents ranges from 95 to 225 per million person years and highest being recorded in Australia and Israel, and lowest in India and Japan.

Ovarian neoplasm accounts for approximately 1% of all malignant tumors in girls aged 17 years or below (Young & Miller, 1975).

Ovarian masses may be of benign or malignant. Dealing with ovarian tumors in adolescents is particularly a challenge because of its rarity, atypical symptoms, malignant potential, and probable serious outcome on the patient's reproductive life.

Functional ovarian cysts are the most common benign ovarian masses detected in adolescents. Amongst all age group, adolescents have the highest rates of PID which pre-disposes them to develop tubo-ovarian abscess. As far as true ovarian tumors are concerned, they

comprise 1.5% of all adolescent tumors with an absolute incidence of approximately 5.5%.

Clinical Presentation:

Ovarian neoplasia are known for their silent presentation and detected mostly as an incidental finding. Commonest presentation will be the complain of only vague abdominal discomfort or bloating sensation which is invariably treated with antispasmodics and antiflatulents. The presenting complaints are pelvic pain, pressure symptoms on bladder or rectum, menstrual irregularities etc. The vast majority of children with malignant ovarian tumors are detected at an advanced stage as in majority of cases it doesn't show symptoms till it reaches advance stage. However it has now become clear that ovarian neoplasms also cause particular symptoms in particular stages of disease progression. Keeping this fact into mind, Symptom index has been developed for screening of ovarian cancer is under trial in USA. Torsion may occur in as many as 35 to 45% of ovarian tumors in adolescents. These cases are wrongly diagnosed as suffering from appendicitis. The use of imaging modalities such as X- ray and ultrasound will increase the diagnostic accuracy to 80%.

According to National Cancer Institute's SEER program, cancer statics review(1973-1988), the most common tumors in

adolescent females (15 -19 years) are germ cell tumors (15%). Dysgerminoma are the most common malignant germ cell tumors, which account for about 30-40% of all ovarian cancers of germ cell origin. Dysgerminoma destroy the ovarian tissue may cause amenorrhoea which must be differentiated from tuberculosis which often presents as an abdominopelvic mass due to encysted ascites.

Endodermal sinus tumor of ovary are also common germ cell tumor seen in paediatric and adolescent age group. They grow rapidly and present usually as abdominal or pelvic pain or abdominal enlargement. Asymptomatic pelvic pain is seen in 100%. Granulosa cell tumors present as precocious puberty in young adolescent or as abnormal vaginal bleeding in the older adolescent. It accounts for about 1-2% of all ovarian malignancies.

Investigation:

Baseline investigation should always include pregnancy test regardless of elicited sexual history. USG, CT Scan, MRI forms the cornerstone for evaluation of the exact size, location, internal consistency, relationship with other structures and also spread to other organs (metastasis). Chest radiograph should be done. Abdominal X-ray for visualizing calcifications in teratomas.

Tumor markers are an important laboratory investigation which are extremely useful in monitoring ovarian neoplasms. CA 125, alphafetoprotein, human chorionic gonadotrophins (HCG) are such tumor markers which are elevated in specific malignancies. CA125 is elevated in epithelial tumors. HCG and alphafetoprotein are raised in endodemal sinus tumor and non gestational choriocarcinoma.

Management:

The main aim of management of ovarian tumor is to conserve the reproductive potential without jeopardizing her life. Asymptomatic cystic adnexal masses requires only observation as these are always benign and regress spontaneously in 3-6 months or promotes regression with oral contraceptives for 3 months.

Surgery is indicated in following conditions:

- Ovarian cyst > 6 cm without regression for 6-8 weeks
- Any cystic lesion > 10 cm
- Solid ovarian lesion
- Ovarian lesion with papillary excrescences in its wall
- Bilateral
- Presence of ascites

Usually cystectomy is the choice of surgery in order to preserve her reproductive potential. Benign conditions should be managed by conservative surgery either by laparoscopy or laparotomy. Surgery like salphingo oopherectomy is consider in cases associated with torsion and infarction.

Endometriosis in adolescent is managed primarily by non surgical methods and includes prolonged suppression of ovulation. Mature cystic teratomas are managed surgically by only removal of the affected gonad with preservation of ipsilateral tube. In case of teratomas, close inspection of the contralateral ovary should be done for evidence of bilateral disease.

Fertility preservation surgeries in adolescent girls:

Preservation of fertility in adolescent girls has been a matter of great concern for the gynaecologist dealing with them, at the risk of ovarian damage. In adolescent girls, fertility may be impaired by repeat ovarian surgeries ,use of gonadotoxic treatment or genetic disorders. In recent times, various options have emerged in order to preserve fertility in teenage girls like cryopreservation of embryos, mature and immature oocytes and ovarian cortex prior to use of gonadotoxic therapy.

Cryopreservation of ovarian tissue has been proposed for adolescent girls suffering from Turner's syndrome, especially the mosaic type.

Treatment of cancer in adolescents should be a multi-disciplinary settings and is based on the degree of malignancy. The primary treatment is surgery with proper staging. In advanced cases of malignant germ cell tumor , which is associated with metastasis, ascites etc surgery is followed by post operative radiation therapy as these tumor are radiosensitive. But the major disadvantage of radiation therapy is the loss of fertility. So, now the treatment of choice is the combination chemotherapy eg. BEP,VBP,VAC.

Adolescents treated for ovarian tumors should be closely followed up, particularly if ovarian preservation was attempted. In the follow up, detailed history, thorough clinical examination, USG and specific tumor marker should be done.

Adolescent Sexuality:

Adolescence is a critical time when there is curosity and increase in sexual drive. This time offer an ideal window of opportunity for building the foundation of sexual and reproductive health in adolescents. The burden of adolescent sexual behavior are an enormous burden on both

adolescent and the society. Developmentally, adolescent reaches physical maturity before they are cognitively able to appreciate the consequence of their behavior. A teenager's major source of information regarding sexuality is his or her peer group, all of whom are experiencing and reinforcing the same behaviors. The family, the major socialize of other behaviors is not as powerful a force in shaping responsible sexual behavior because of parental discomfort in sex education and sexual discussion. A healthy attitude towards healthy sexual interaction, and awareness of their behavior and needs, followed by counseling can prevent many traumatic consequences.

MATERIALS AND METHODS

A total of 150 cases of adolescent girls aged 13-19 years who attended gynaecological OPD and emergency department in ISO & KGH hospital, Triplicane were included in the study.

Study Design:

Prospective study.

Inclusion Criteria:

Married / unmarried, non –pregnant, non lactating adolescent girls aged 13-19 years.

Exclusion criteria:

Teenage pregnancy

History:

Detailed history with regard to the gynaecological problems was taken from the patient and girl's mother was also interviewed to get accurate details about any previous medical problems if present.

Physical Examination:

A thorough clinical examination including height, weight, secondary sexual character, general examination of breast, thyroid, cardiovascular system, respiratory, and central nervous system or any congenital anomalies were noted.

Body mass index (BMI) or quetelet index was calculated using the formula:-

BMI =
$$\frac{\text{Weight in kg}}{\text{(Height in cm)}^2}$$

General examination comprising thyroid examination, presence of hirsutism, pubertal stage of breast and pubic hair were graded according to Marshall and Tanner staging. External genitalia examined. Per abdominal examination done to rule out any mass. Per rectal and bimanual pelvic examination done whenever indicated.

Then patients were subjected to various investigations like haemogram, coagulation profile, hormonal assays, and ultrasonography depending on diagnosis clinically.

Amenorrhoea cases were referred to endocrinologist and genetic counseling done. For all cases, relevant investigation like karyotyping was done and the diagnosis confirmed. Patients were also referred to haematologist, oncologist in those indicated.

OBSERVATIONS

Table 1: Age Distribution

Age in years	No. of patients	Percent
13	11	7.3
14	12	8.0
15	21	14.0
16	18	12.0
17	35	23.3
18	25	16.6
19	28	18.6
Total	150	100.0

The table shows the age distribution in the study group. It ranged from 13-19 years of age. Maximum number of adolescent girls attending the gnaecology OPD in KGH were of 17 years of age (3/150), followed by girls with age of 19 years (28/150).

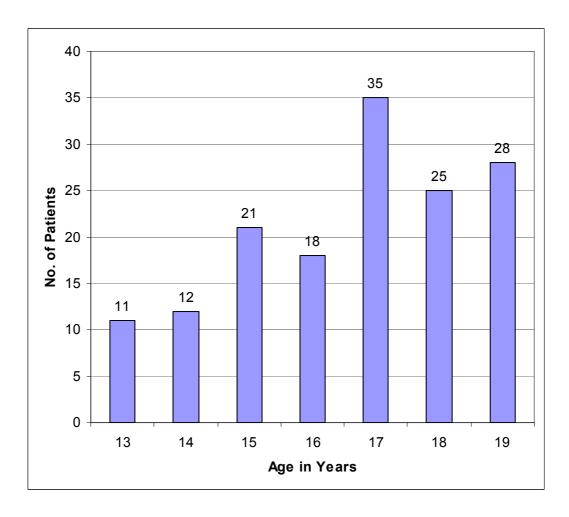


Chart 1: Age Distribution

This bar diagram shows the diagramatic representation of age distribution among the adolescent girls in the study group. Maximum incidence is of 17 years.

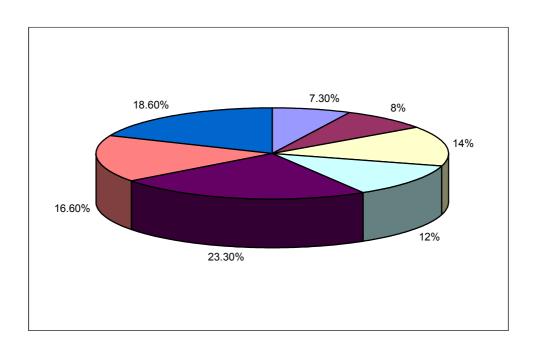


Chart 1: Age Distribution

The bar chart depicts that the, adolescent girls of age group 17 years attending Gynaecology OPD were maximum (23.3%).

Table 2: Socioeconomic status

Socioeconomic Status	No. of Patients	Percent
III	21	14.9
IV	80	53.3
V	49	32.6
Total	150	100.0

In the present study, all the girls were mostly from family with lower socio economic status either from Class-III / IV / V.

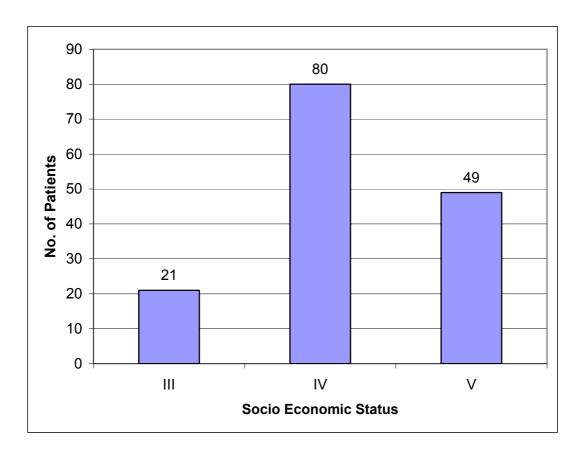


Chart 2: Socioeconomic status

In the present study majority of adolescent girls came from low socio economic background with highest number of 80 cases under Class-IV socio economic status (80/150).

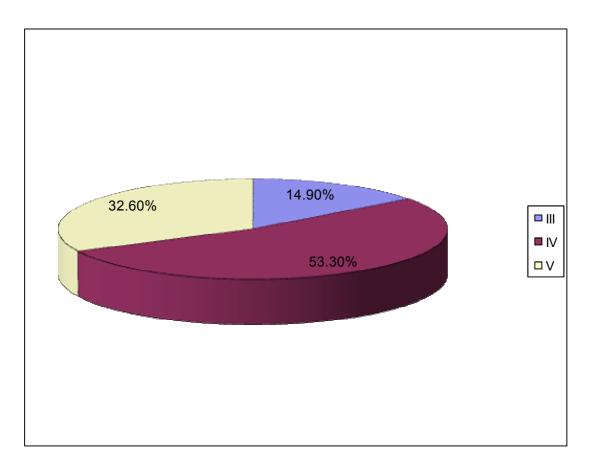


Chart 2 : Socioeconomic status

Out of 150 adolescent girls, 14.9% belonged to class III socio economic status, 53.3% belonged to class IV, 32.6% belonged to class V socio economic status.

Table 3: Educational Status

Education	No. of patients	Percent
Primary	19	12.6
Secondary	80	53.3
Higher secondary	51	34.0
Total	150	100.0

This table shows the number of adolescent girls in the study group with their educational status. Though literacy rate among the adolescent girls was found to be 100 percent, but percentage of school dropouts were 3.33% (5/150).

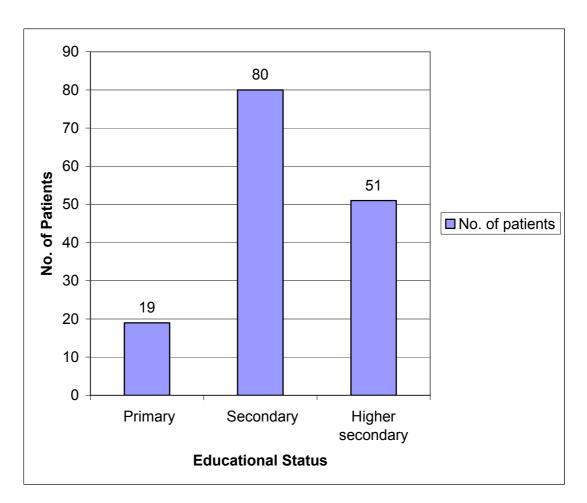


Chart 3: Educational status

In the present study, majority of girls were studying in secondary standard (80/150), followed by girls with higher secondary educational status.

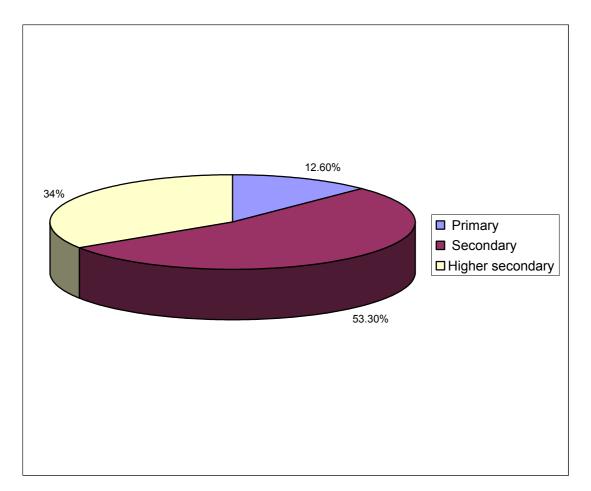


Chart 3: Educational status

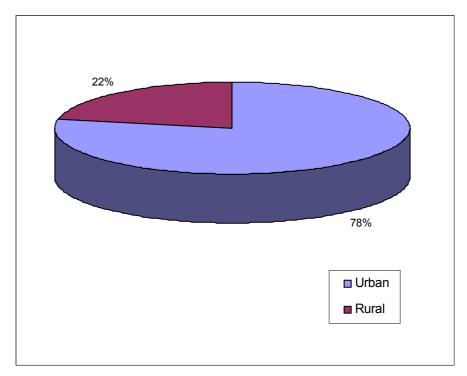
This pie chart represents that maximum percentage (53.3%) of adolescent girls in the present study group were having educational status as secondary standard.

Table 4: Demography

Place of living	No. of patients	Percent
Urban	117	78.0
Rural	33	22.0
Total	150	100.0

Majority of the adolescent girls under study belonged to urban area (78.0%)

Chart 4: Demography



This pie chart shows that maximum percent (78.0%) of adolescent girls in the study group came from the urban population.

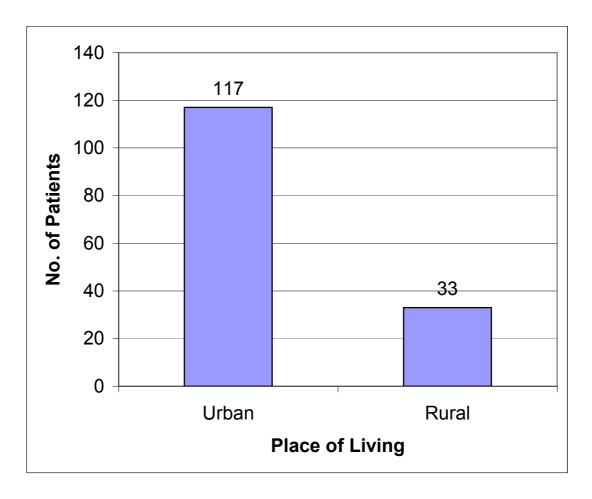


Chart 4: Demography

Out of 150 cases studied, 117 girls came from the urban area and rest of 33 girls were from rural area in and around of KGH, Chennai.

Table 5: Body mass index (BMI)

BMI	No. of patients	Percent
Under weight(≤19)	39	26.0%
Normal (20-24)	84	56.0%
Overweight (25-29)	22	14.6%
Obese (30-34)	3	2.1%
Morbid obesity (≥35)	2	1.3%
Total	150	100.0%

In the study group, adolescent girls were categorized into normal, underweight, overweight, obese and morbidly obese, as per their BMI calculated using weight measured in Kg and height in cm.

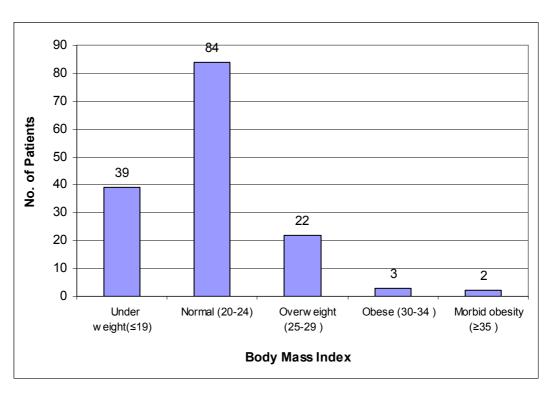


Chart 5: Body mass index (BMI)

Out of total 150 cases of adolescent girls, majority (84/150) were having normal BMI, followed by (39/150) underweight, overweight (22/150). There was only few cases of obesity (3) and morbid obesity (2).

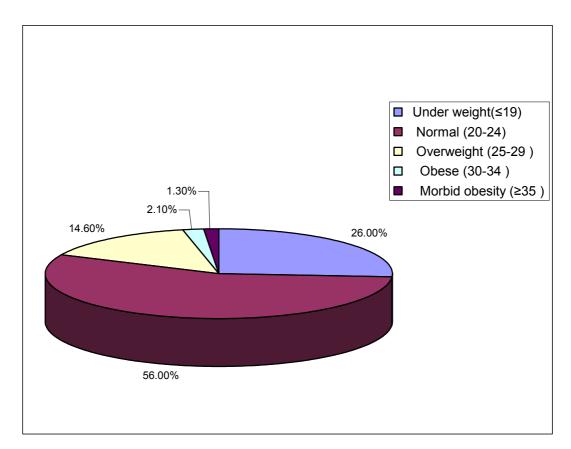


Chart 5: Body mass index (BMI)

Among the 150 adolescent girls in the study group, maximum percent of girls had normal BMI (56.0%), 17.6% had higher range of BMI while about 26% were underweight.

Table 6: Anaemia incidence (Hb% estimation)

Anaemia	No. of Patients	Percent
Severe	4	2.6
Moderate	80	53.3
Mild	38	25.3
Normal	28	18.6
Total	150	100.0

Majority of the adolescent girls under study were anaemic, which ranged from mild to severe type based on the haemoglobin estimation.

- 28/150 cases had no anaemia with hb% ≥ 12 gm%.
- 38/150 cases had mild anaemia with hb% ranging from11-11.9 gm%.
- 80/150 cases had moderate anemia with hb% ranging from 8.1-10.9 gm%.
- 4/150 cases had severe anaemia hb% <8 gm%

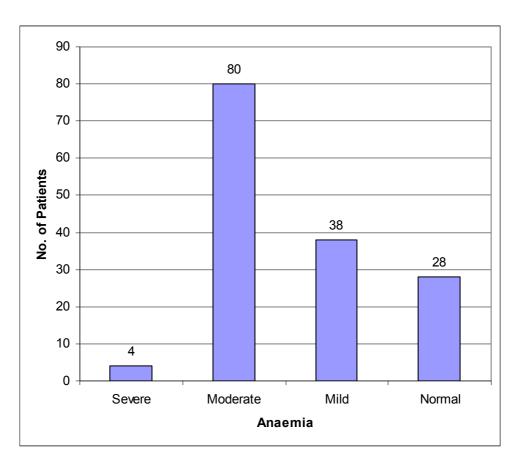


Chart 6: Anaemia incidence (Hb% estimation)

Moderate type of anemia with haemoglobin percent ranging from 8.1-10.9 gm% were most prevalent among the study group.

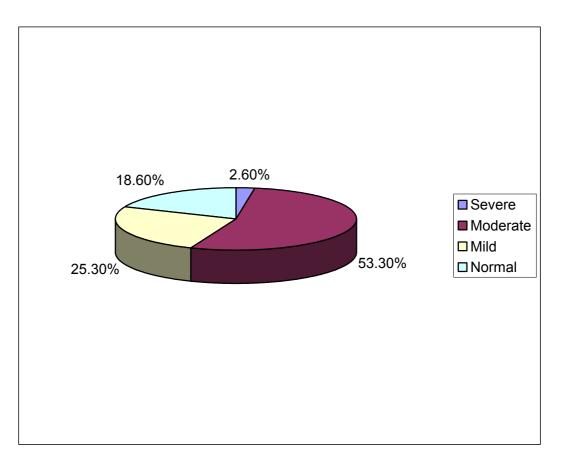


Chart 6: Anaemia incidence (Hb% estimation)

Majority of the adolescent girls under study were anaemic, 25.3% had mild anaemia, 53.3% (maximum) had moderate anaemia and 2.6% suffered from severe anaemia. Only 18.6% of cases were not anaemic as per the Hb% estimation.

Table 7: Presenting complaints in the study population

Complaints	No. of Patients	Percent
Menstrual complaints	147	59.2%
Vaginal discharge	51	20.5%
Lower abdominal pain	14	5.64%
Urinary problem	15	6.04%
Weight problem	14	5.64%
Others	7	2.82%
Total	248	100.0%

In the present study, various presenting complaints among the adolescent girls were depicted in the table. Most common presenting complaints among the teenage girls was menstrual complaint. Majority of girls were having one or more morbidity.

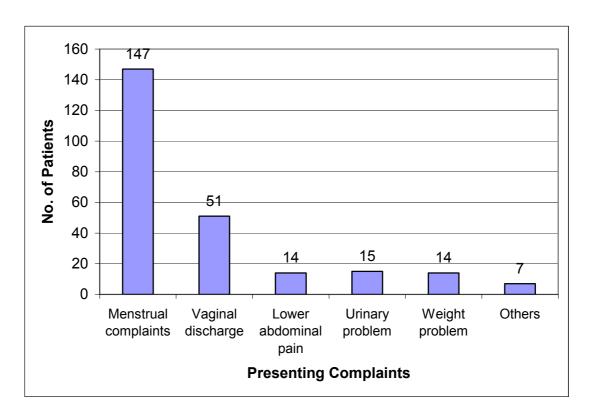


Chart 7: Presenting complaints in the study population

In the present study, girls were allowed to have multiple responses regarding their presenting complaints. Menstrual complaints the most common complaint (147) among the study group attending the OPD, followed by complaint of white discharge p/v (51).

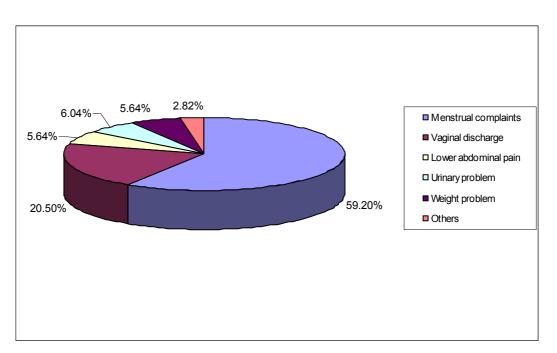


Chart 7: Presenting complaints in the study population

Majority of the girls had menstrual complaints 59.2%, followed by second common complaint was vaginal discharge 20.5% and then followed by urinary tract infection 6.04%.

Table 8 : Types of menstrual dysfunction

Menstrual disorders	No. of patients	Percent
Amenorrhoea	9	6.12
Menorrhagia	32	19.04
Oligomenorrhoea	81	55.1
Hypomenorrhoea	1	0.68
Dysmenorrhoea	24	16.3
Total	147	100.0

Menstrual complaints being the most common complaints among the studied adolescent group of girls. Menstrual complaints ranged from amenorrhoea to menorrhagia. Dysmenorrhoea was also included in this category.

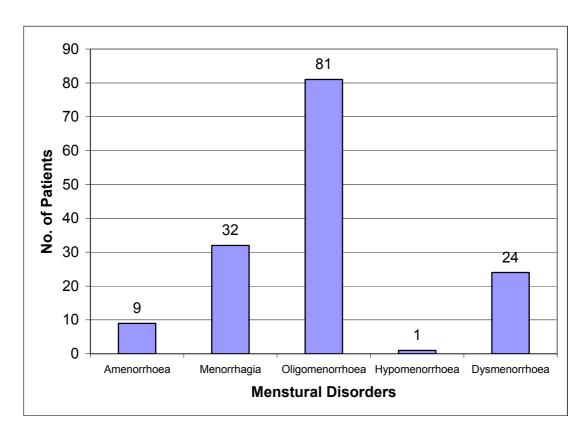


Chart 8: Types of menstrual dysfunction

Among the menstrual complaints, the most common type of complaint was that of oligomenorrhoea (81/147), followed by menorrhagia.

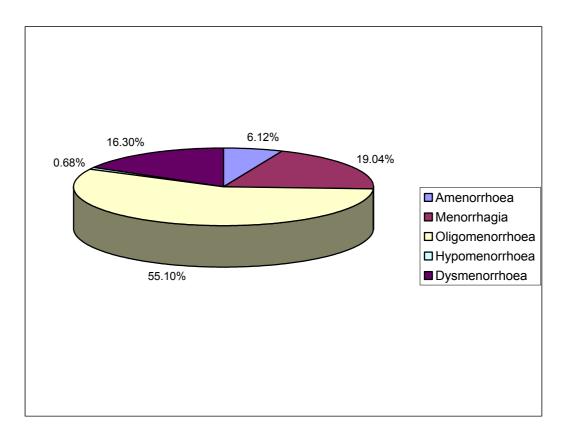


Chart 8: Types of menstrual dysfunction

Majority of girls were suffering from oligomenorrhoea (55.1%) followed by those presenting with menorrhagia (19.04%)

Table 9: Association between BMI and PCOS among the study group with menstrual complaints.

BMI	No. of patients	Percent	Chi-square Value	P Value
Under Weight	1	3.6%		
Normal	14	50.0%		
Over Weight	8	28.6%	28.294	0.000
Obese	3	10.7%	28.294	0.000
Morbid Obesity	2	7.1%		
Total	28	100.00%		

A highly significant statistical association between BMI and PCOS was seen. Adolescent having higher BMI had increased incidence of PCOS in the USG.

Chart 9: Association between BMI and PCOS among the study group with menstrual complaints

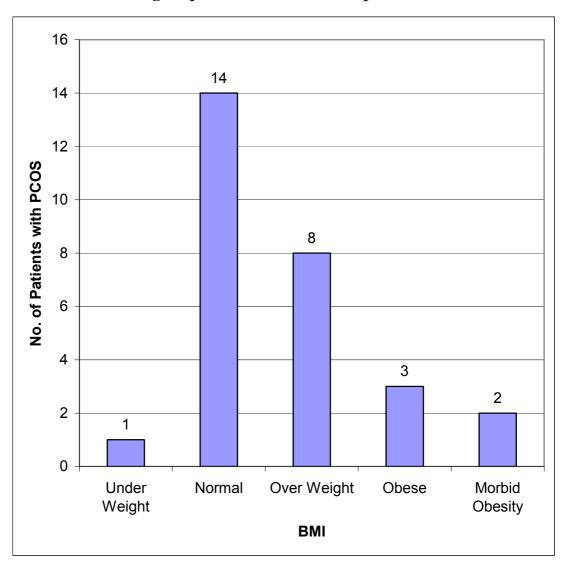


Table 10 : Etiology of Amenorrhoea

Etiology	Karyotype	No. of patients	Percent
Mullerian agenesis	46 XX	4	44.4
Turner syndrome	45 X0	2	22.2
Turner mosaic	45 X0 / 46 XX	1	11.1
Constitutional delay	46 XX	1	11.1
Pure gonadal dysgenesis	46 XX	1	11.1
Total		9	100.0

Commonest cause of primary amenorrhoea in this study group were Mullerian agenesis (44.4%).

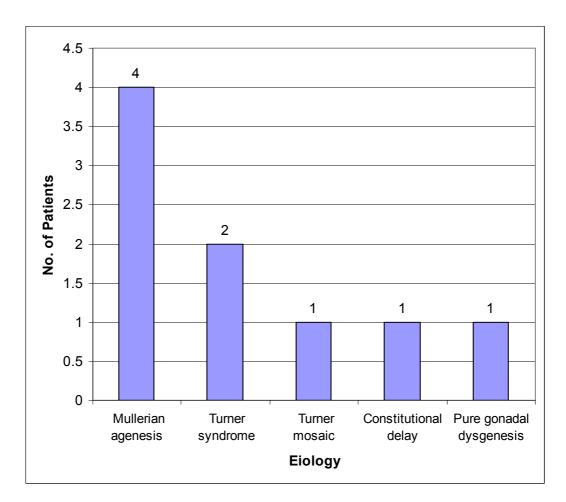


Chart 10: Etiology of Amenorrhoea

Out of 9 cases giadnosed with primary amenorrhoea in the study group.the commonest cause was MRKH syndrome.

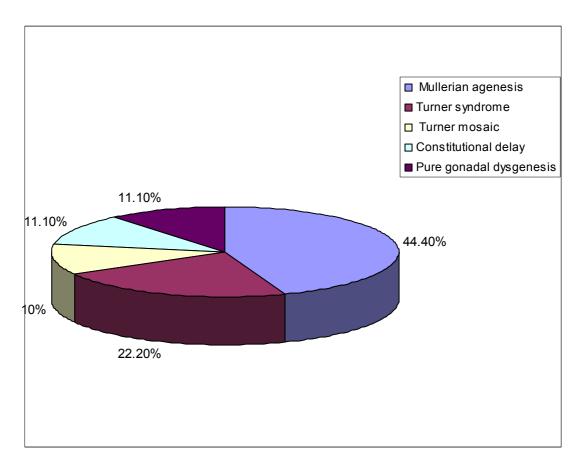


Chart 10: Etiology of Amenorrhoea

In the present study, maximum percent (44.4%) of cases amenorrhoea was diagnosed with mullerian agenesis, followed by Turner's syndrome (22.2%).

Table 11: Etiology of Menstrual dysfunctions

Etiology	No. of patients	Percent
Dysfunctional uterine bleeding	86	74.7
PCOS	24	20.8
Thyroid disorder	4	3.47
Hyperproactinemia	1	0.86
Total	115	100.0

This table shows various causes for the menstrual irregularities in the adolescent girls under study. Most common is the Dysfunctional uterine bleeding.

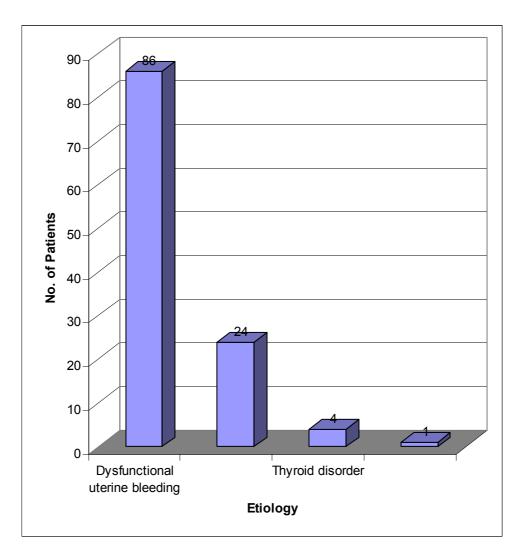


Table 11: Etiology of Menstrual dysfunctions

Out of the total 115 case of menstrual dysfunction, 86 cases were due to dysfunctional uterine bleeding without any pelvic pathology followed by PCOS (24) which was the second most common cause for the menstrual disorder in the adolescent age group under study.

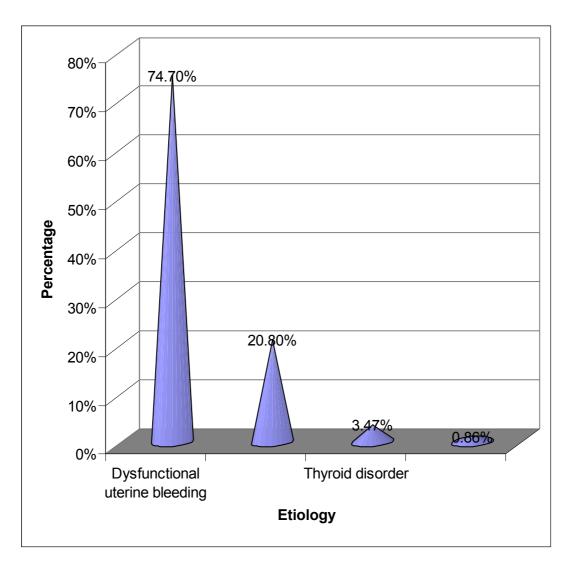


Table 11: Etiology of Menstrual dysfunctions

Commonest cause of menstrual dysfunction in the adolescent girls under study was dysfunctional uterine bleeding without any pelvic pathology (74.4%).

Table 11: Age in years and dysmenorrhoea in adolescent girls

Age group in years	Total no of cases	No. of cases with dysmenorrhoea
13-15 years	42	6
16-19 years	99	1 8
Total	141	24

In the present study, the prevalence of dysmenorrhoea is higher in the adolescent girls of age group between 16-19 years.

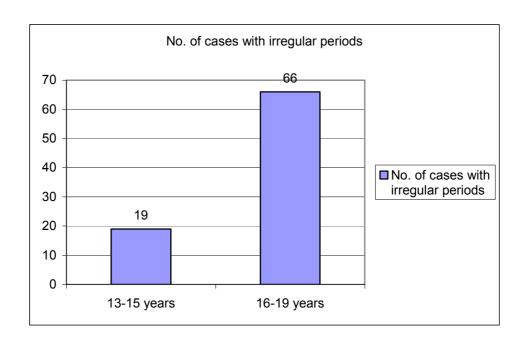
Table 12 : Age Group in years and Irregular Periods in Adolescent

Girls

Age group in years	No. of cases with irregular periods	% within age group in years	% within irregular periods count	Chi square value	P-value
13-15 years	19	45.2%	22.4%	5.656	0.017
16-19 years	66	77.6%	66.7%	3.030	0.017

There is a significant statistical association between the age group in years with complain of irregular periods.

Chart 12: Age in years and irregular menstrual complaints in adolescent girls



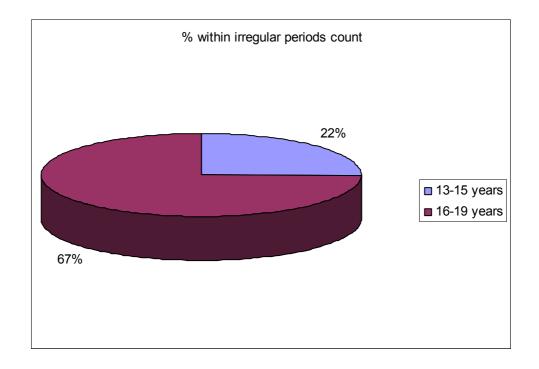
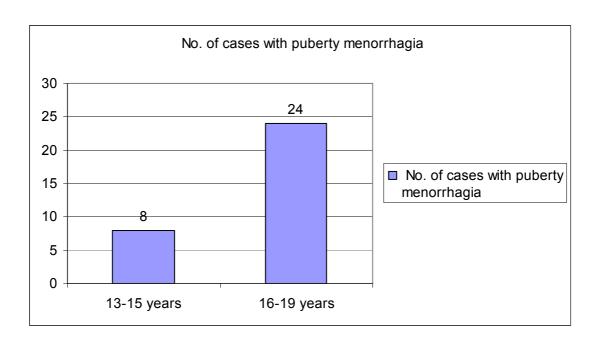


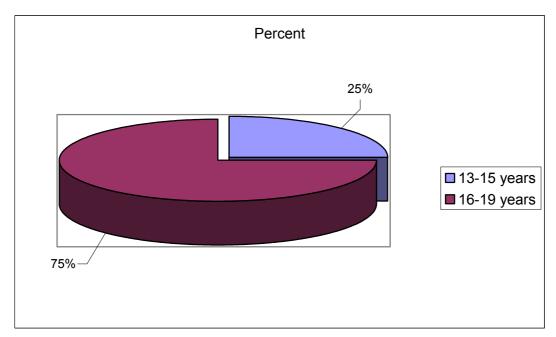
Table 13: Age in years and Puberty menorrhagia

Age group in years	No. of cases with puberty menorrhagia	Percent	Chi square value	P- value
13-14 years	8	25%		
16-19 years	24	75.0%	8.000	0.005
Total	32	100%		

There is a significant statistical association between age group in years and puberty Menorrhagia.

Chart 13: Age group in years and Puberty menorrhagia in adolescent girls under study.





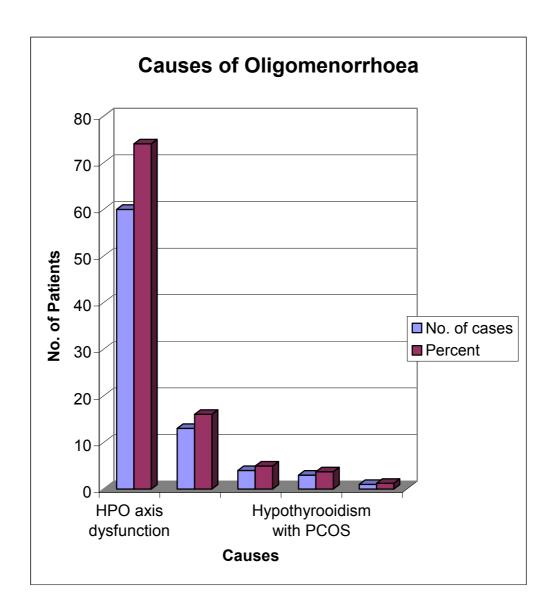
This chart shows that puberty menorrhagia were more common in age group of 16- 19 years i.e; 24 out of all 32cases of puberty menorrhagia.

Table 14: Causes of oligomenorrhoea

Cause	No. of cases	Percent
HPO axis dysfunction	60	74.07
PCOS	13	16.04
Hypothyroidism	4	4.93
Hypothyrooidism with PCOS	3	3.70
Hyperprolactinemia	1	1.23
Total	81	100.0

In the present study 81 cases of oligomenorrhoea were studied.

Commonest cause of oligomenorrhoea was HPO axis dysfunction 74.07%, followed by PCOS accounting for 16.04%.



In the present study , the main cause for oligomenorrhoea among the study group of adolescent girls was the immaturity of hypothalamus – pituitary-ovarian axis (74.7%).

Table 15: Association between anaemia and puberty menorrhagia

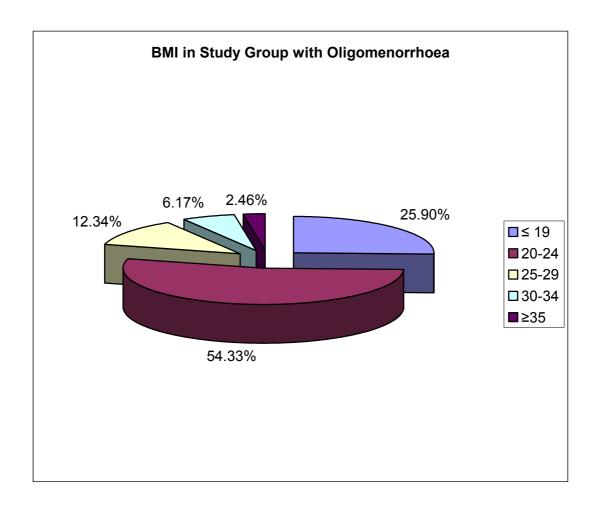
Anaemia	No of cases with menorrhagia	Chi square value	P-value
Normal	1		
Mild	2		
Moderate	28	66.750	0.000
Severe	1		
Total	32		

There is highly significant association between the anemia status and the puberty menorrhagia.

Table 16: BMI in the study group and oligomenorrhoea

BMI	No. of cases	Percent
≤ 19	20	25.9%
20-24	44	54.325%
25-29	10	12.34%
30-34	5	6.17%
≥35	2	2.46%
Total	81	100.0

Out of 81 cases of oligomenorrhoea 44 cases had normal BMI, 20 were underweight and 17 had BMI higher than the normal.



In the present study 54.3% of cases with oligomenorrhoea had normal BMI, 25.9% had were underweight and 20.9% had BMI higher than normal.

Table 15: Ovarian tumors in the study group

Tumors	No. of cases	Percent
Ovarian malignancy	2	40.0%
Twisted ovarian cyst	1	20.0%
Simple serous cyst	2	40.0%
Total	5	100.0%

In this study, 5 adolescent girls were diagnosed with ovarian tumor (benign or malignant). Most common type of ovarian tumor found in the present study was simple serous ovarian cysts (2/5). Out of the two malignant ovarian tumor, 1 case was of Juvenile granulose cell tumor fig. 1(a) and 1(b) and other was Yolk sac tumor fig.2(a) and 2(b).

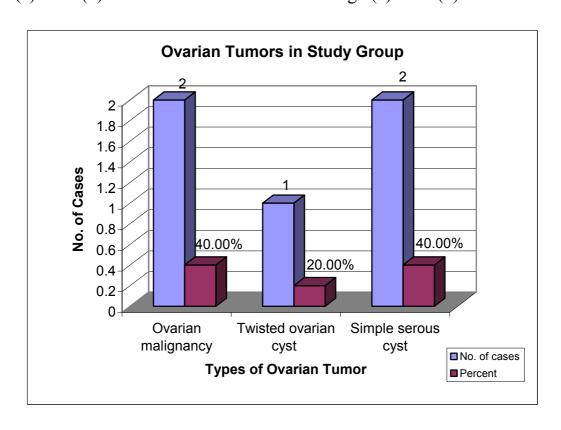




Fig. Gross image of Juvenile Granulosa Cell Tumour of ovary

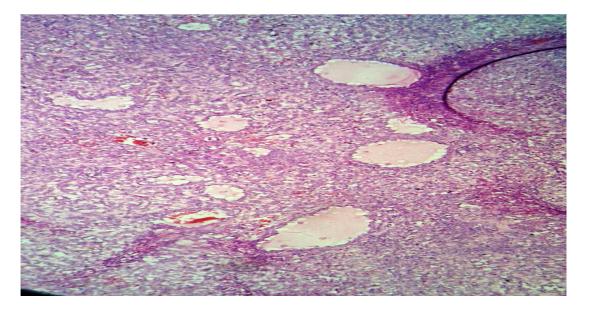


Fig: Microscopic image

Many follicles are lined by cells with atypical 'hobnail' nuclei simulating a tubulocystic clear cell arcinoma

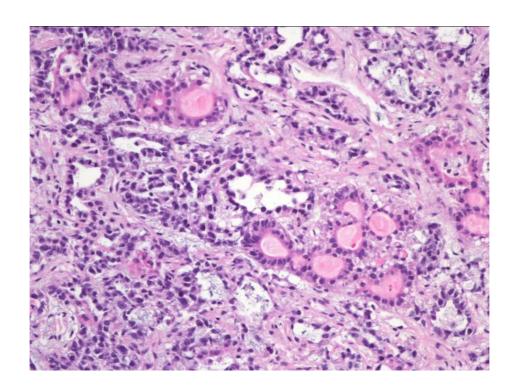


Fig 2a: Yolk sac tumour of ovary

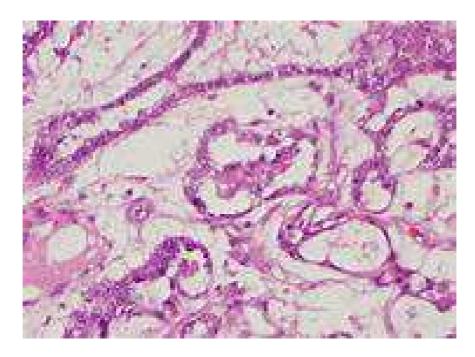


Fig 2(b): Yolk sac tumour of ovary with Schiller duval bodies

DISCUSSION

This study was conducted at ISO & Government Kasturba Gandhi Hospital for Women & Children to analyse the various gynaecological problems prevalent among the adolescent girls and to know various cause and factors associated with it.

In the present study, maximum incidence of gynaecological problems was seen in the age group of 17 years (23.3%). About 3.33% of girls were school drop outs. Majority of them belonged to family of Class IV socio-economic status and 78.0% came from urban areas.

Disturbances of menstruation, either physiological or pathological, are the most common presenting complaint in adolescent gynaecological clinic. The present study shows that menstrual disorders are the most common gynaecological dysfunction among the adolescent girls (62.3%). These ranges from amenorrhoea to menorrhagia.

1) Primary Amenorrhoea:

In present study amenorrhoea accounted for 4.28% (9/150) of cases, in comparison to that reported by **Sebanti et al (6.45 %) and study by Archana** *et al* (6.25%). Out of the total of 9 cases of Primary amenorrhoea, four cases was found to be of diagnosis Mullerian dysgenesis. Two cases were diagnosed as Turner syndrome. One case underwent vaginoplasty.

Mashchak et al., 1981, Shearman and Roberts et al., 1982, Sebanti et al., 2005 showed that the commonest cause for primary menorrhea was mullerian agenesis (MRKH syndrome).

In the present study, the commonest cause of of primary amenorrhoea with normal secondary sexual characteristics was mullerian agenesis (MRKH syndrome) 33.3%. Common cause of primary amenorrhoea with delayed secondary sexual characteristics were (Garden et al., 1998, Edmond et al., 1999)

- i) Turner syndrome
- ii) Gonadotrophin deficiency
- iii) Constitutional delay

In present study, Turner syndrome is the commonest cause of primary amenorrhoea with delayed secondary sexual character.

2) Oligomenorrhoea:

Oligomenorrhoea is the most common menstrual complaint in the present study (57.4%). **Morgan and London** *et al.*, 1993 showed that the most common cause of oligomenorrhoea is HPO axis followed by causes like PCOS, stress, thyroid disorder etc. In the present study, HPO axis dysfunction was the most common cause of oligomenorrhoea accounting for 74.04%

Prevalence of menstrual disorders in various studies

Study group	Percent
Balasubramanian et al, 2000	26.62%
Goswami et al , 2005	58.8%
Kulkarni et al , 2011	11.16%
Present study	55.1%

PCOS (20.8%) is the second most common cause of oligomenorrhoea followed by hypothyroidism (3.47%). PCOS which was diagnosed based on the clinical criteria of menstrual dysfunction, hyperandrogenism and ultrasonographic findings.

There were 14/24 cases of PCOS who presented with complain of oligomenorrhoea and 10 cases presented with menorrhagia. PCOS is the cause of menstrual irregularities in 24/114 (21.0%) cases in the present study which is comparable to the finding of 22.9% in the study conducted by **Archana** *et al* (2013).

In the present study, the most least common cause of menstrual irregularities is polymenorrhoea (0.9%).

3) Puberty menorrhagia:

Puberty menorrhagia accounts for 19.8% of all menstrual complains in the present study. 10 out of 28 cases were admitted in our hospital and 4 cases had severe anaemia and 2 moderate anaemia, which required blood transfusion.

Most of cases of puberty menorrhagia had anaemia ranging from mild to severe, predominantly anemia ranging from haemoglobin percentage of 8.1- 10.9 gm%. Anaemic patients were treated with haematinics both oral and parenteral preparation, hormones and blood transfusion wherever indicated.

4) Dysmenorrhoea:

Primary dysmenorrhoea was presenting complaint in 10 cases out of total 18 girls in the present study group. In rest of 8 cases, dysmenorrhoea was an associated complain with other complains of menstrual irregularities .3 out of 10 cases had problem severe enough to prevent them from going to school. These girls on detailed history elicitation, their mother and sibling also had similar history of severe dysmenorrhoea. **Dickens** *et al.*, also mentioned family history of primary dysmenorrhoea.

Prevalence of dysmenorrhoea was high in late adolescent age group. Balasubramanian et al., 2000 and Rehman et al., 2004 also reported similar study findings.

5) Leucorrhoea:

In this study, 51 cases (21.07%) of vaginal discharge was the second most commonest complaint in the adolescent girls. Most of them had physiological leucorrhoea which responded to counseling and maintenance of personal hygiene.

6) Ovarian tumor:

Ovarian tumor were found in 5/150 cases (3.3%). Out of these 2 had simple serous cysts and 1 with twisted ovarian cyst. There were 2 cases of malignant ovarian tumour which turned out to be a case of Juvenile granulosa cell tumor and Yolk sac tumor confirmed by histopathological studies. **Sebanti** *et al.*, 2005 and **Archana** *et al.*, 2013 have reported a higher incidence of ovarian tumor in their series, 15.3% and 4.5% respectively.

Study group	Incidence %
Oumachigui et al.	6%
Dhamne et al.	2.25%
Huggmen et al.	1%
Sebanti et al.	15.3%
Archana et al.	4.5%
Present study	3.3%

Two cases in the study presented with bartholin cyst, out of which one case was of recurrent bartholin cysts turning to bartholin abscess, was treated by drainage and antibiotic. Another case presented with injury to perineum, due to sexual assault.

SUMMARY

- Low socio economic group coming from urban population was the maximum group of girls studied.
- Maximum incidence of gynaecological problems was seen in the age group of 17 years.
- Among the various presenting complaints in studied adolescent girls, menstrual irregularities were the most common.
- Oligomenorrhoea was the most common menstrual problem in this study.
- Anovulation is the most common cause of menorrrhagia in adolescents.
- Majority of teenage (81.2%) suffered from anemia ranging from mild to severe types of anaemia.
- 21.42% patients with puberty menorrhagia received blood transfusion.
- The most commonest cause of oligomenorrhoea was the Hypothalamic -pituitary – gonadal axis dysfunction.

- Counseling and reassurance was an integral part of treatment strategies.
- PCOS and hypothyroidism were the other endocrinological abnormalities common among the adolescent girls.
- Dysmenorrhoea was more common among the late adolescent age group.
- Leucorrhoea in adolescent girls in the present study were most commonly physiological, counseled and explained to maintain proper hygiene.
- MRKH Syndrome (Mullerian agenesis) is the commonest cause of primary amenorrhoea.
- A combined effort of team comprising gynaecologist,
 endocrinologist, genetist is required in arriving the final diagnosis
 of in cases of primary amenorrhoea.
- In the present study benign ovarian tumor were more common than malignant ovarian tumor.

CONCLUSION

Adolescent gynaecology is an important sub- specialized part of gynaecology. The importance of the reproductive health problems among the adolescent has emerged tremendously during recent years.

Endocrinal abnormalities like PCOS are associated with future reproductive as well as metabolic morbidity. Therefore, it is very important to evaluate and manage endocrinal abnormalities in adolescent properly so as to secure a healthy reproductive life.

Menorrhagia, at all stages of life severely affects the quality of life. Effective management of puberty menorrhagia is mandatory in order to avoid adolescent with anaemia. As mentioned earlier, adolescent girls being the direct reproducer of future generation, it is important to avoid adolescent embarking on pregnancy with anaemia, which in long run reduce perinatal and maternal morbidity and mortality.

Teenage problems need to be dealt with utmost sensitivity. Counseling teenage girls as well as parents is an integral part of the treatment strategies. Awareness regarding health, nutrition and hygiene should be included in the counseling in order to curtail problems like anemia, leucorrhoea.

Though, adolescent gynaecology is not a new subject. But, it still needs greater attention and increased awareness, in order to protect and promote the health of adolescents. As the problems are specific to this age group, setting up of a separate 'Adolescent Gynaecological Clinics' is the need of the hour.

BIBLIOGRAPHY

- 1. ACOG committee opinion: the initial reproductive health visit 2010
- 2. Age Data- Census of India 2011.
- 3. Balsubramanian P 2000. Health needs of poor unmarried adolescent girls. A community based study in rural Tamil Nadu. A draft paper from Rural Women's Social Education Centre, Tamil Nadu.
- 4. Current diagnosis and treatment, obstetrics and gynaecology, 10th edition chapter 56, 926:929.
- 5. Current evaluation of amenorrhoea, Fertil steril 2006;86:S148
- Dickens A. Excessive Menstrual Bleeding and Dysmenorrhoea.
 Clinical Obstetric and Gynaecology 1974; 17:665-659.
- 7. Edmunds D.K. Primary amenorrhoea, Progress in Obstetrics and Gynaecology (Vol.10) John Studd: 281-1993.
- 8. Franks S. Medical Progress article: Polycystic ovary syndrome. N
 Engl J Med 1995;301-25.

- 9. Fujimoto Y, Miller J.H, Klein N A, Michael R, : Am J. Obst. Gyn. 177: 1419;1997
- 10. Gharanin, Wther Worth DM. Battyental association of the steroid synthesis gene CYP1la with polycystic ovary syndrome and hyperandrogenism. Hum Mol Genet 1997;6:397-402.
- 11. Goswami Sebanti et al. A Profile Adolescent girls with Gynaecological Problems. J Obstetric Gynaecology India 2005; 55(4): 353-355.
- 12. Guzick DS, Wing R, Smith D, Berga SL, Winters SJ. Endocine consequences of weight loss in obese, hyperandrogenic women before and after weight loss. J Clin Endocrinol Metab 1989;68:173-79.
- 13. Holte J, Berne T, Berne C, etal. Restored insulin sensitivity but persistently increased early insulin secretion after weight loss in obese women with polycystic ovary syndrome. J Clin Endocrinol Metab 1995;80:2586-93
- 14. Jeffcoate's principles of gynaecology, 8th edition, 2008;621
- Justin S Bromn, Gaery L Warne. Growth in precocious puberty.
 Krishna U, Patwardhan M. Evaluation and Management of Primary

- Amenorrhoea FOGSI Publication. The Adolescent girl (Editors Vinita Salvi, Usha Krishna) 57-1993.
- 16. Krishna U,Parulekar S, Salvi V(Editors). The Adolescent Girl FOGSI Publication 1991.
- Kumari Archana et al. Adolescent Gynaecological Problems : A
 Clinical Study. J of Evolution of Medical and Dental Services.2013.
- 18. Leon Speroff, Merca Fritz. Clinical Gynaecologic Endocrinology and Infertility 8th ed. 2011:384-87.
- 19. Marshall WA, Tanner JM. Variation in pattern of pubertal changes in girls. Arch Dis Child 1969; 44:291-303.
- 20. Mashchak, C.A., Kletzky O.A., Davajan V. and Mishell D.R.1981. Clinical and Laboratory evaluation of patients with primary amenorrhoea obstet. Gynaecol. 57: 715-721
- 21. Michael Kappy, Thomas Stuart, Alvin Perelman, Robert Clemons, et al. Children'Health Centre of St. Joseph's Hospital(MK,TS), and Phoenix Children's Hospital(AP,RC),
- 22. Phoenix, Arizona 85013. Suppression of Gonadotropin Secretion by a long acting Gonadotropin Releasing Hormone

- Analogue(Leuprolide Acetate, Lupron Depot) in Children with Precocious Puberty.
- 23. Miller H.G., Cain VS, Rogers S.M., Gribble J.N: Fam. Plan. Perspect: 31:4, 1999.
- O. Sheil, M Turner. Adolescent Gynaecology. Progress in Obstetrics and Gynaecology; 12:251-233
- 25. Reindollar RH, Byrd JR, McDonough PG. Delayed Sexual development: A Study of 252 patients. AM J Obstet Gynaecol 1981;140-371
- 26. Richard H, Reindollar, M.D. and Paul. G. Mc Donough, M.D.Adolescent Menstrual Disorders. Clinical Obst. & Gynaecol. Vol.26, No.3, September 1983
- 27. Revised 2003 consensus on diagnostic criteria and long term health risks related to polycystic ovary syndrome (PCOS).
- 28. Walter Worth DM, Bennetist, Gharani, et al. Linkage and association of insulin gene VNTR regulatory polymorphism with polycystic ovary syndrome. Lancet1997;349:986-89

- 29. Shearman, R.P and Roberts J. 1982. The embryology and andocrinology of primary amenorrhoea a study of 140 patients. Clin. Reprod. Fert. 1:117-130.
- 30. Sexual and Reproductive health of younger adolescents, WHO-2011; Page 7-9

PROFORMA

Name	:		
Address	:		
Age	:		
OP/IP No.	:		
Height	:	Weight:	BMI:
Educational Status	:		
Occupation	:		
Marital Status	:		
MENSTRUAL HISTO	RY	:	
• Age of Menarche			
• LMP			
• Duration of Cycles	S		
Amount of Bleeding	ng		
• Number of Pads /	Clothes used per	day	
 Associated Compl 	aints		
CHIEF COMPLAINTS	S	:	
(A) Menstrual Abnormal	ities	:	
 Amenorrhoea 	:		
 Menorrhagia 			
 Oligomenorrhoea 			
 Polymenorrhoea 			
• Hypomenorrhoea,	etc		

- (B) Dysmenorrhoea
- (C) Lower Abdominal Pain
- (D) Leucorrhoea
- (E) Abdominal Mass
- (F) Pre Menstrual Syndrome
- (G) Others

GENERAL EXAMINATION

- Thyroid Examination
- Breast Examination
- Secondary Sexual Characters
- CVS
- RS

CLINICAL EXAMINATION

- P/A
- P/V
- P/R

INVESTIGATIONS

ULTRASOUND

PROVISIONAL DIAGNOSIS

ABBREVIATION

WHO – World Health Organisation

GH – Growth Hormone

IGF-I – Insulin like growth factor-1

FSH – Follicular stimulating hormone

LH – Luteinising hormone

TSH – Thyroid stimulating hormone

FT₃ – Free Tri iodothyronine

FT₄ – Free Tetraiodothyronine

GnRH – Gonadotropin Releasing Hormone

DHEA-S – Dehydroandroepisterone-sulfate

VNTR – Variable number tandem repeat

PCOS – Polycystic Ovarian Syndrome

USG – Ultrasonography

E₂ – Estradiol

LPD – Luteal Phase Defect

OCP – Oral Contraceptive Pills

PT – Prothrombin Time

ApTT – Partial Thromboplastin Time

BEP – Bleomycin, Etoposide,

VBC – Vincristine, Bleomycin, Cisplatin

VAC - Vinblastin, actinomycin, cyclophosphamide

INFORMATION SHEET

- We are conducting a study on "STUDY ON ADOLESCENT GYNAECOLOGICAL PROBLEMS" among patients attending Kasturba Gandhi Government Hospital Chennai and for that your clinical details may be valuable to us.
- We are selecting certain patients and if you are found eligible, we may be using your clinical details in such a way so as to not affect your final report or management.
- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.
- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator	Signature of participant
Date:	Signature of parent (if minor)

CONSENT FORM

STUDY TITLE : STUDY ON ADOLESCENT GYNAECOLOGICAL PROBLEMS

STUDY CENTRE :	Institute of Socia	al Obstetrics,	
	Govt. Kasturba Ga	ndhi Hospital	l,
	Chennai-5.		
PARTICIPANT NAME :	A	GE:	O.P.NO./ I.P NO.
opportunity to ask the questisfaction. I understand to withdraw at any time without I understand that in need my permission to loo further research that may understand that my identity published, unless as require arise from the study.	estion and all my of that my participation but giving any reason investigator, regulated by at my health recomble conducted in relay will not be reveal ed under the law. I	questions and on in the studen. ory authorities ords both in lation to it, evolution any information agree not to	tedure for the above study, I have the doubts have been answered to my by is voluntary and that I am free to es and the ethics committee will not respect to the current study and any ven if I withdraw from the study. I formation released to third parties of restrict the use of any or results that
Signature of Investigator:			Place :
Study Investigators Name		·	Institution
Signature / Thumb Impress	sion of patient		
Signature of parent(if mino	or)		

INSTITUTIONAL ETHICS COMMITTEE MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013 Telephone No: 044 25305301 Fax: 044 25363970

CERTIFICATE OF APPROVAL

To

Dr.Radhe Akang,

Postgraduate in Obstetrics & Gynaecology, Kasthurba Gandhi Govt. General Hospital, Madras Medical College, Chennai-5.

Dear **Dr.Radhe Akang**,

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal on Adolescent Gynaecological Problems" "Study entitled No.29032014.

The following members of Ethics Committee were present in the meeting held on 11.03.2014 conducted at Madras Medical College, Chennai-3.

Dr. C.Rajendran, M.D, 1.

-- Chairperson

2. Dr.R.Vimala, MD.,

4.

7.

-- Deputy Chairperson

Dean, MMC, Ch-3.

-- Member Secretary

Prof. Kalaiselvi, M.D, Vice Principal, MMC, Ch-3

Prof. Nandhini, M.D,

Inst. of Pharmacology, MMC, Ch-3

Prof.Bhavani Sankar, M.S, 5.

-- Member

-- Member

Prof & HOD General Surgery, MMC, Ch-3

Prof.V.Padmavathi, M.D,

-- Member

I/c. Director of Pathology, MMC, Ch-3

Thiru. S. Govindasamy, BA., BL -- Lawyer

8. Tmt.Arnold Saulina, MA MSW -- Social Scientist

Thiru.S.Ramesh Kumar,

-- Lay Person

Administrative Officer, MMC, Ch-3.

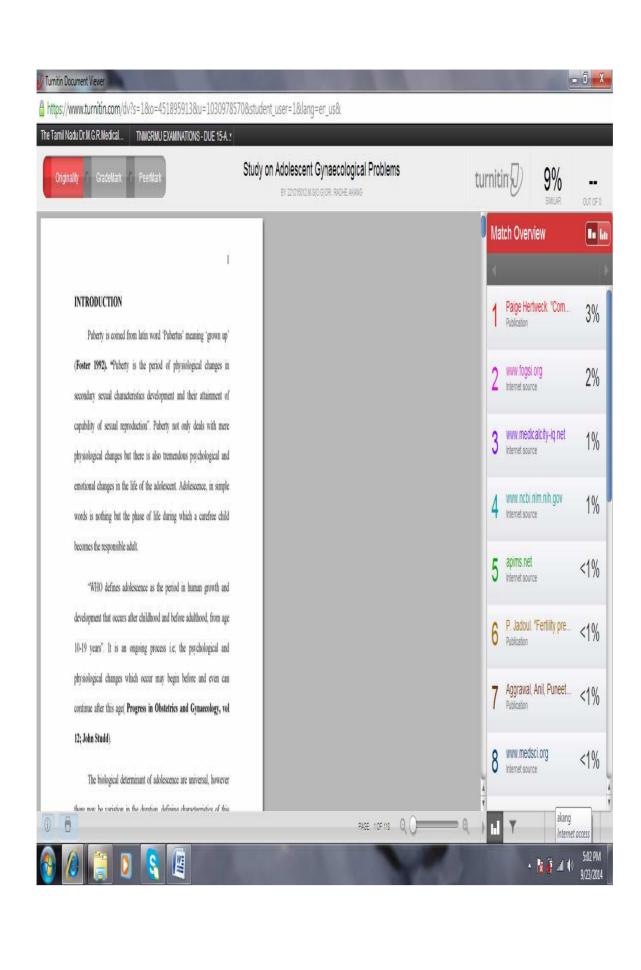
We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

> Member Secretary, Ethics Committee TITUTIONAL ETHICS COMMIT

> > # 1413114



o.No	NAME	ONdido	AGE	AGE_G	SES	EDN	LOCALITY	HT	WT	BMI	Com.1	Com.2	Com.3	Com.4	Com.5	Com.6	Com.7	Com.8	Com.9	Com 0	GOITRE	HIRSUTIS	HB	XRAY	NSG	TSH	PL	H	DIAGNOSI	CAUSE
1.	Kavitha	107,065	17	2	3	2	2	147	36	1	1								1		2	2	3	1	1	1	1	1	2	1
2.	Bharathi	180,515	19	2	4	3	2	147	36	1	1								1		2	2	3	1	1	1	1	1	2	1
3.	Banu	175,880	17	2	4	3	2	152	44	2	1								1		2	1	2	1	1	1	1	1	2	1
4.	Monisha	118,091	14	1	4	2	2	145	44	2	1										2	2	2	1	1	1	1	3	2	1
5.	Rohini	113,082	18	2	5	3	2	160	68	2	1	1							1		2	2	2	1	1	1	1	3	2	1
6. 7.	Priya	24,734	17	2	3	2	2	156	63	3			1						1		1	2	2	1	2	1	1	3	2	_1
8.	Vanishri	11,350	16	2	5	2	2	145	55	5	1	1				1					2	2	2	1	2	1	1	2	2	3
9.	Anandhi	127,560	17	2	5	2	2	152	48	2				1					1		2	2	4	3	0	1	1	0	5	0
10.	Zeenat		17	2	4	2	2	152	48	2		1									2	2	2	1	1	1	1	3	2	_1
11.	Iswarya	137,690	17	2	4	2	2	150	43	2	1	1									2	2	2	1	1	1	1	3	2	1
12.	Pavitra	145,209	17	2	5	2	2	152	48		1			1							2	2	2	1	1	1	1	3	2	1
13.	Ashwini		16	2	5	2		147	42	2		1		1							2	2	2	1	2	1	1	2	2	3
14.	Shakila	t - ' - t	15	1	3	2		148		3			1						1		1	1	2	1	1	2	1	1	2	2
15.	Sangeeta		15	1	3	2		148		1			1			1			1		1	2	2	1	1	1	1	1	3	1
16.	Jayanthi	, i	17	2	5	3		157	66	3	1		1						1		1	2	2	1	2	1	1	2	3	3
17.	Sneha	t - ' - t	13	1	4	1		147	36	1				1							2	2	3		0		1	0	5	0
18.	Bhavani	134,209		2	4	2		155		1				1							2	2	4	1	1	1	1	1	2	1
19.	Ramya	, i	17	2	4	2		155		1	1			1							2	2	3		1	1	1	1	3	1
20.	Divya	t - ' - t	19	2	4	3		148		2											1	2	4	1	2	1	1	1	3	3
21.	Shanthi		19	2	4	3				3	1										2	1	2	1	2	1	1	2	3	
22.	Surya	, i	15	1	5	2		160		1	_			1							2	2	3		1	1	1	0	5	0
23.	Deepa	t - ' - t	19	2	5	3		148		2				1							2		3		1	1	1	1	2	1
24.	Ishrat		18	2	5	3		148		2				1							2		2		1	2	1	3	2	1
25.	Ashwini		16 18	2	3	3		144 152		3	1					1					1	2 1	2	1	1	1	1	3	2	1
26.	Sowmya		17	2		2				2	- 1	1		1		- 1					2	2		1	2 1	1	1	2 1	2	3
27.	Kowsalya Suguna	201,367		2	3	2		148 148			1	1		- 1							2		2		1	1	1	1	3	2 1
28.	Mary	201,367		2	4	2		155				1									2		2		1	1	1	1	3	
29.	Firtosh	153,289			5	3		148				_							1		1	2	4		1	1	1	1	2	
30.	Tenmozhi	195,604		2	5	3		149			1	1									2		3		1	1	1	1	2	<u> </u>
31.	Sulekha	153,729			5	2		152				_							1		1	2	3		2		1	2	2	3
32.	Pooja	115,024			4	3		155							1				ġ		2		4		3		1	0		
33.	Sneha	164,523		1	4	2		155				1									2		3		1	1	1	3	2	1
34.	Madhavi	172,094			4	2		145						1	1						2		3				1	0		0
35.	Kalaivani	152,703		1	4	2		148						1							2		2				1	0		
36.	Srinithi	151,638		1	4	2		148				1									2		1	3		2	1	2	2	3
37.	Sonia	181,626		2	5	3		145						1		1				1	2		3			0	1	0		
38.	Gayathri	146,288		1	4	2		146						1	1						2		4				1	0		
39.	Subatha	181,420		2	3	3		149			1			1							2		4	1	1	1	1	1	2	1
40.	Iswarya	178,390		1	4	2		150				1						1			1	1	2	1	1	2	1	2	3	2
41.	Preethi	172,653		1	4	2		147				1									2	2	2		1	1	1	1	2	
42.	Hemavati	182,749		2	5	3		155		1	1			1							2		2		1	1	1	1	2	1
43.	Tenmozhi	192,731			4	3		144		2	1			1							2	2	2	1	1	1	1	3	2	1
44.	Puspa	193,762	17	2	5	3	2	153	41	1	1			1							2	2	2	1	1	1	1	3	2	1
45.	Nandini	201,735	17	2	4	2	1	148	40	2	1	1									2	1	1	1	2	1	1	2	3	3

																														П	\neg
S.No		NAME	ONdidO	AGE	AGE_G	SES	EDN	LOCALITY	HT	WT	BMI	Com.1	Com.2	Com.3	Com.4	Com.5	Com.6	Com.7	Com.8	Com.9	Com 0	GOITRE	HIRSUTIS	HB	XRAY	nse	TSH	PL	Η	DIAGNOSI	CAUSE
	46.	Kavitha	218,301	15	1	5	2	1	145	52	2			1								2	2	2	1	1	1	1	1	4	1
	17	Malathi		19		3	3		144					1						1		1	1	2		2	2	1	1	3	3
	40	Anitha		18		4	1		148		2			1						1		1	2	2	1	1	1	1	3		1
	40	Manjula	203,641		2	3	2		147											1		1	1	3	1	1	1	1	3		1
	50	Viji	216,720			4	2		147		1	1				1						2	2	3		1	1	1	3		1
	51.	Nazreen	183,920	19	2	4	3	2	147	42	2	1				1						2	2	3	1	1	1	1	3	2	1
	52.	Vani	190,367	19	2	4	3	2	150	43	2										1	2	2	2	3	1	1	1	0	6	7
		Iswarya	200,012	13	1	4	2	1	144	40	2	1								1		1	2	3	1	1	1	1	3	2	1
		Surya	172,293	17	2	4	2	2	150	43	2	1			1							2	2	3	3	1	1	1	3	2	1
	55.	Rekha	172,923	18	2	4	3	2	148	40	2				1							2	2	4	3	1	1	1	0	5	0
		Kalairasi	160,279	13	1	4	2	1	142	36	1				1							2	2	4	3	1	1	1	0	5	0
		Beula	182,630	13	1	5	1	1	147	36	1	1				1						2	1	2	1	2	1	1	2	2	3
	58.	Lavanya	215,261	15	1	5	2	2	144	40	2	1				1						2	2	2	1	1	1	1	1	2	1
		Ratna	202,435	18	2	5	3	2	150	43	2	1				1						2	2	3	1	1	1	1	1	3	1
		Divya	209,817	13	1	5	2	1	142	36	1				1	1						2	2	3	3	1	1	1	0	5	0
		Sangitha	207,639	15	1	4	2	2	147	42	2	1		1								1	2	2	1	0	1	1	3	3	1
		Gomathy	182,773	18	2	5	1	2	144	40	2				1					1		1	2	3	3	1	1	1	0	5	0
		Karpagam	170,274	17	2	4	3	2	147	42	2	1	1									2	2	2	1	1	1	1	3	2	1
	64.	Usha	172,927	19	2	4	3	2	155	70	4	1		1			1					2	1	2	1	1	2	1	2	2	3
		Divya	187,209	19	2	4	3	2	147	42	2	1			1							2	2	2	1	1	2	1	1	2	3
		Soundarya	187,735	13	1	3	1	2	147	42	2			1	1							2	2	3	1	1	1	1	3	2	1
		Sujatha	198,364	19	2	4	3	1	155	70	4	1			1		1			1		2	2	4	3	1	1	1	3	2	1
		Karpagam	193,024	17	2	4	2	2	147	42	2	1	1									2	2	3	1	1	1	2	1	2	5
	70	Rajlaxmi	210,936	18	2	5	3	2	155	44	1				1					1		2	2	3	3	0	1	1	0	5	0
	71	Monisha		19		5	3		154		2				1							2	2	3	3	0	1	1	0	5	0
	70	Sonia		16		5	2		155		2				1							2	2	2	3	0		1	0		0
	73	Susmitha	218,392			3	2		147	42	2			1								2	1	2	1	1	1	1	1	2	3
	74.	Monisha	204,662			3	2		148						1							2	2			1	1	1	1		_1
	75	Jamuna	209,374			4	3		145					1								2	2	2	1	1	1	1	1	2	_1
	76	Sonia	196,381			4	2		150						1							2	2	2		1	1	1	3		1
	77	Monica	194,465				1		144						1							1	2			1	1	1	3		1
	70	Nandini	198,273			4	3		147													1	2	2		1	1	1	3		1
	70	Anitha	201,982			4	2		147			1										1	2	2		1	1	1	3		_1
	80	Pavithra	218,221			3	2		150					1						1		2	2			1	1	1	3		_1
	81	Vaisnavi	219,301			4	2		144						1	_					4	2	2					1	0		-0
	82	Bhavani Priva	210,372			4	3 1		152						1	1	1				1	2	2	3		3		1	0		7
	02	Priya Vinodini	201,836				2		144 150						1					1		1	2	2		2		1	2		3
	Ω/Ι					4	2								1							2	2					1	0		0
	O.F.	Chandra Shobana	214,253 212,837			5 5	3		146 155						1							2	2		3			1	0		0
	86.		213,840			5 5	<u>ა</u> 1		155													2	2			1	1	1	1	2	1
	87.	Laxmi Meena	217,003			5 5	3		145						1							1	2	4	1	0		1	1		1
	00	Selvi	218,324				3		150						- 1		1			1		2	1	2		2		1	1		3
	89.	Jannat	217,820			5	2		144							1				- 1		2	2	2		1	1	1	1	2	1
	90.	Vidya	219,032			5	2		146						1							2	2	2				1	0		0
Ь		viuya	213,032	14	لالنا	J			1+0	JU					1								2		J	U	_ '			J	U

S.No		NAME	ONdIdO	AGE	AGE_G	SES	EDN	LOCALITY	HT	WT	BMI	Com.1	Com.2	Com.3	Com.4	Com.5	Com.6	Com.7	Com.8	Com.9	Com 0	GOITRE	HIRSUTIS	HB	XRAY	nse	TSH	PL	H	DIAGNOSI	CAUSE
		Nithya	208,936	19	2	4	3	2	147	36	1			1								2	2	2	1	1	1	1	3	2	1
		Kavitha	207,593	17	2	4	2	2	152	44	2			1								2	2	2	1	1	1	1	3	2	1
		Swathi	213,750	15	1	4	2	2	147	36	1			1								2	2	2	1	1	1	1	3	2	1
		Kavitha	209,375	19	2	3	3	2	147	36	1			1								2	2	2	1	1	1	1	1	2	1
		Nivetha	216,354	14	1	3	2	2	147	42	2			1								2	2	2	1	2	1	1	2	2	3
		Arulmothi	218,364	16	2	4	2	1	147	42	2					1						2	2	3	1	1	1	1	0	6	7
	97.	Latha	214,673	17	2	4	2		145		2			1	1							2	2	2	1	1	1	1	1	3	1
	98. 99.	Ayesha	216,480	19	2	5	1	2	147	42	2			1								2	2	2	1	1	1	1	3	3	1
		Janani	219,333	19	2	4	3	2	147	58	4			1			1					2	2	2	1	2	1	1	1	2	3
	101	Sandhya	215,730	16	2	5	2	2	155	70	3	1			1							2	1	2	1	2	2	1	2	3	3
	101.	Thilaga	215,300	16	2	5	2	2	155	67	3	1			1		1					2	2	2	1	2	2	1	2	3	3
	102	Meena	215,361	17	2	4	1		147	36	1	1										2	2	4	1	1	1	1	1	3	1
	103.	Sasirekha	214,772	18	2	5	1		147	42	2				1							2	2	4	1	1	1	1	3	3	1
	105.	Lilly	215,801	14	1	4	2		144		2											2	2	2	1	1	1	1	3	3	1
	106.	Uma	215,623	14	1	4	2		147	42	2	1	1									2	2	2	1	1	1	1	3	3	1
	107	Geetha	230,281	18	2	5	3		162	38	1	1										2	2	3		0		1	3	2	1
	108	Manjula		18	2	4	3		155		1		1			1		1				2	1	2	1	1	1	1	3	3	1
	109.	Suraya	<u> </u>		2	3	2		144	40	2				1							2	2	1	1	1	1	1	1	3	1
	110	Gayathri	220,934	17	2	4	2		144	40	2											2	2	2	1	1	1	1	1	3	1
	111	Kamatchi			1	4	2		155		4	1		_			1			1		2	1	1	1	1	1	1	1	2	4
	112.	Vanaja	220,193		2	4	3		155		2			1								2	2	2	1	2	2	1	1	2	1
	113	Jothi	230,647	14	1	4	2		157	62	3			1								1	2	2		2	2	1	1	1	1
	111	Rosy Radhika	235,180 246,751	17	2	4	2 1		162 162		1			1								1 2	2	2	1	1	1	1	3	3	1
	115.	Nithya	224,132	17	2	4	1		155		2			1								2	2	2	1	1	1	1	3	3	1
	116.	Saraswati	234,521	15	1	3	1		155		2		1	'				1	1			1	1	4	1	1	1	1	1	3	2
	117.	Sujatha	230,012		1	3	2		147		2		-	1				-	_			2	2	2				1	1	2	1
	440	Kumari	248,321			5	2		147					1								2	2	2		2		1	3		
	110	Deepa	241,979			5	2		148					•								2	2			2		1	1	3	
	400	Sharmila	248,723		2	4	3		147													2	2	2		1	1	1	3		
	121.	Sophi	210,294			4	2		146						1							2	2	3		3		1	2	2	3
	122	Shanthi	221,902			4	3		155													2	2			1	1	1	1	2	
	122	Shylaja	231,562			4	3		155				1									2	2		1	1	1	1	1	2	1
	101	Harini	240,961			4	2		146				1									2	2		1	1	1	1	1	2	1
	105	Devaki	245,371			5	2		152													2	2			1	1	1	1	2	
	126.	Devi	259,102			5	2		155			1			1							2	2	2		1	1	1	1	2	1
	127.	Tulasi	251,833			5	3		154			1										1	2	2		1	1	1	3		1
	120	Jotilaxmi	260,012			5	3		154													1	2	4	1	1	1	1	3		1
	120	Raji	275,201			5	3		150								1			1		1	1	2		2	1	1		2	3
	120	Sumathy	278,192			5	2	2	155	52	2	1			1							2	2	2	1	1	1	1	3	2	1
	131.	Ramya	279,012			4	3		145													2	2	3	1	1	1	1	3	2	1
	132.	Banu	282,362	16		3	2	1	155	48	1				1							2	2	2	3	0	1	1	0	5	0
	133.	Sindu	270,912	16	2	3	2	2	147	40	2				1							2	2	3	3	0	1	1	0	5	0
	134.	Divya	256,091	19	2	4	1	2	145	55	3	1			1							2	2	4	1	1	1	1	1	2	1
	135.	Kamatchi	245,612	19	2	5	3	2	153	55	2	1								1		1	1	2	1	2	1	1	1	2	3

S.No		NAME	ONdidO	AGE	AGE_G	SES	EDN	LOCALITY	HT	WT	BMI	Com.1	Com.2	Com.3	Com.4	Com.5	Com.6	Com.7	Com.8	Com.9	Com 0	GOITRE	HIRSUTIS	HB	XRAY	nse	TSH	PL	LH	DIAGNOSI	CAUSE
	136.	Vasugi	260,456	19	2	4	3	2	153	41	1	1										1	2	4	1	1	1	1	1	2	1
	137.	Renuka	271,891	18	2	5	3	1	155	48	1	1	1									2	2	2	1	1	1	1	1	2	1
	138.	Nithya	289,012	18	2	5	3	1	153	41	1	1	1									2	2	2	1	1	1	1	1	2	1
	139.	Rani	281,282	18	2	4	1	2	149	38	2		1									2	2	4	1	1	1	1	3	2	1
	140.	Priyadarsini	290,128	13	1	4	1	2	147	40	2	1		1		1					1	2	2	3	1	3	1	1	0	6	7
	141.	Susila	290,123	14	1	5	2	2	146	50	2				1							2	2	3	3	0	1	1	0	5	0

_		~		
1.	Age	Group	ın	vears

 $1 \rightarrow 13 - 15$ years

 $2 \rightarrow 16 - 19$ years

2. Socio economic Status

 $3 \rightarrow \text{Class III}$

 $4 \rightarrow \text{Class IV}$

 $5 \rightarrow \text{Class V}$

3. Educational status

 $1 \rightarrow Primary$

 $2 \rightarrow Secondary$

 $3 \rightarrow$ Higher Secondary

4. Localities

 $1 \rightarrow Urban$

 $2 \rightarrow Rural$

5. Body Mass Index (BMI)

 $1 \rightarrow \leq 19$ (Under weight)

 $2 \rightarrow 20 - 24$ (Normal)

 $3 \rightarrow 25 - 29$ (over weight)

 $4 \rightarrow 30 - 34$ (Obese)

 $5 \rightarrow \geq 35$ (Morbid obesity)

6. Complaints

1 - Irregular Periods

2 - Scanty Periods

3 - Profuse prolonged bleedings P/v

4 - White discharge p/v

5 - lower abdominal pain

6 - Excessive weight gain

7 - increased hair growth over face

8 - Neck swelling

9 - Dysmenorrhoea

7. Goitre

1 - Present

2 - Absent

8. Hirsution

 $1 \rightarrow Present$

 $2 \rightarrow Absent$

9. Hb% $1 \rightarrow \text{Normal} (\geq 12 \text{ gm }\%)$

 $2 \rightarrow Mild (11.0 - 11.0 gm \%)$

 $3 \rightarrow Moderate (8.1 - 10.9)$

 $4 \rightarrow \text{Severe} (< 9 \text{ gm }\%)$

10. USG $0 \rightarrow \text{Not done}$

 $1 \rightarrow Normal$

 $2 \rightarrow PCOS$

 $3 \rightarrow \text{T.O. Mass}$

11. TSH $1 \rightarrow Normal$

 $2 \rightarrow High$

12. PL $1 \rightarrow Normal$

 $2 \rightarrow \text{Not done}$

 $0 \rightarrow High$

13. LH $1 \rightarrow Normal$

 $2 \rightarrow High$

14. Diagnosis $1 \rightarrow \text{Oligomenorrhoea}$

 $2 \rightarrow$ Menorrhagia

 $3 \rightarrow PCOS$

 $4 \rightarrow Polymenorrhoea$

 $5 \rightarrow \text{Leucorrhoea}$

 $6 \rightarrow Others$

15. Cause $0 \rightarrow \text{Leucorrhoea}$

 $1 \rightarrow HPO$ axis dysfunction

 $2 \rightarrow$ Hypothyroidism

 $3 \rightarrow PCOS$

4 → Polymenorrhoea

 $5 \rightarrow$ Hyperprolactinaemia

 $6 \rightarrow$ Thyroditis

 $7 \rightarrow Others$

sl no.	Name	IP/OP no.	Age	SE	EDU	U/R	Ht	Wt	ВМІ	Arm span	Consan	Compl	Breast	Ax. Hair	Pub hair	som Abn	Hirstut	Ext gen	Vag	Uterus	Ovaries	met abn	TSH	K-typ	Dx	Cause
1	Gomathy	10385	18	IV	S	U	160	58	22.1	140	Non	1	IV	2	2	NIL	NIL	1	3	3	1	NIL	1.85	46XX	Α	3
2	Sowmya	34289	15	IV	Р	U	132	51	29.27	120	II	1	I	3	3	Р	NIL	2	2	1	3	NIL	3.9	45X0	Α	1
3	Puspalata	45342	17	IV	S	U	146	40	18.77	142	Non	1	II	3	3	NIL	NIL	2	2	1	3	NIL	1.925	46XX	Α	5
4	Ayesha	37890	17	IV	S	U	135	34	18.66	138	Non	1	II	3	1	NIL	NIL	1	1	1	1	NIL	3.01	46XX	Α	4
5	priyadarshini	43890	16	IV	S	U	132	39	22.38	130	Non	1	I	3	3	Р	NIL	2	2	2	3	NIL	0.75	45X0,46XX	Α	2
6	jenifer	12546	18	٧	S	U	145	33	15.7	140	III	1	I	2	2	NIL	NIL	1	3	4	1	NIL	1.9	46XX	Α	3
7	Akila	490045	15	IV	S	U	142	33	16.37	140	II	1	Α	3	3	NIL	NIL	2	1	3	1	NIL	1.75	46XX	Α	3
8	Komala	23471	18	٧	Р	R	160	44	15.97	155	Non	1	III	2	2	Р	NIL	2	2	2	2	NIL	1.867	45X0	Α	1
9	Sathya	223100	16	IV	S	U	142	34		140	III	1	ı	2	2	NIL	NIL	2	3	4	1	NIL	1.254	46XX	Α	3

Age in years

- 1. Ht Height in CM
- 2. Wt Weight in Kg
- 3. Arm arm Span in CMS
- 4. Cons Consanguinity
- 5. Complain 1 Primary Amenorrhoea
- 6. Axl. Hair 1 Absent, 2 Normal, 3 Sparse
- 7. Pubic Hair
- 8. SAB Somatic Abnormalities
- 9. Ex. Gen External Genitalia 1 Normal, 2 Infantile
- 10. Vagina 1 Normal, 2 Infantile, 3 Dimple
- 11. Uterus 1 Normal, 2 Hypoplastic, 3 Rudimentary, 4 Absent
- 12. Met. Abn. Matabolic Abnormalities
- 13. USG Ultra Sonographic
- 14. Ovary 1 Normal, 2 Streak, 3 Absent
- 15. SES Socio Economic Status
- 16. Education: U Uneducated, P Primary, S Secondary, H Higher Secondary
- 17. Locality: U Urban, R Rual
- 18. Diagnosis: A Primary Amenorrhoea
- 19. Cause: 1 Turner Syndrome, 2 Turner Mosaic, 3 MRKH (Mullerian Agenesis),
 - 4 Constitutional Delay, 5 Pure Gonadal Dysgenesis.