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Original Research Article

Etiological Spectrum and Systematic Evaluation of Primary Amenorrhea in Adolescents

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Abstract:

Background: Primary amenorrhea is an important clinical condition in adolescent gynecology that indicates possible abnormalities in the hypothalamic–pituitary–ovarian axis, reproductive anatomy, or systemic health. Early identification of the underlying cause is essential for appropriate management and prevention of long-term complications.

Aim: To evaluate the etiological spectrum and clinical, hormonal, and radiological characteristics of adolescents presenting with primary amenorrhea.

Methodology: A hospital-based observational cross-sectional study was conducted in the Department of Obstetrics and Gynaecology at Nalanda Medical College and Hospital, Patna, India, over nine months. A total of 29 adolescents aged 14–18 years with primary amenorrhea were included. Data were collected through detailed history, clinical examination, anthropometric assessment, hormonal evaluation (FSH, LH, estradiol, prolactin), TSH and pelvic ultrasonography. Statistical analysis was performed using SPSS version 25.

Results: Normogonadotropic hypogonadism was the most common diagnosis (48.3%), followed by hypergonadotropic hypogonadism (31.0%) and hypogonadotropic hypogonadism (20.7%). Auxological parameters showed no significant differences among groups, while hormonal levels demonstrated significant variation. FSH and LH were markedly elevated in hypergonadotropic hypogonadism, whereas estradiol levels were highest in normogonadotropic cases.

Conclusion: Primary amenorrhea in adolescents shows diverse etiologies, with normogonadotropic hypogonadism being the most frequent. A systematic approach combining clinical evaluation, hormonal analysis, and imaging is essential for accurate diagnosis and appropriate management.

Keywords: Primary amenorrhea, adolescents, hypogonadism, etiological spectrum, hormonal evaluation, reproductive disorders.

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Introduction

Primary amenorrhea (PA) refers to failure to menstruate by the age of 14 years in the absence of secondary sexual characteristics or failure to menstruate by the age of 16 years whether secondary sexual characteristics are present or not. It is a clinical issue of significance in pediatric endocrinology and adolescent gynecology since normal menstruation is an important predictor of hormonal and reproductive well-being. Menarche is considered an important milestone in the development of the female puberty, which is an indication of the active work of the hypothalamic-pituitary-ovarian (HPO) axis, normal anatomical development of the reproductive tract, and sufficient systemic health. Failure of menstruation to occur within the normal age range is one of the issues that should be critically considered in order to determine underlying etiological factors and prescribe commendable management [1].

Primary amenorrhea is comparatively uncommon in comparison to secondary amenorrhea, but its clinical importance is enormous as the number of potential underlying causes is very high. The disease can be caused by dysfunctions of the hypothalamus, pituitary gland or ovaries, reproductive tract, systemic diseases and genetic defects. There is the need to identify early and do regular reviews since certain etiologies are linked with long-term reproductive

effects, endocrine as well as psychological effects. Also, some of the causes might need prompt treatment to avoid additional complications like infertility, osteoporosis or metabolic issues [2].

Among adults, gonadal failure, central hypogonadism, and mullerian agenesis are the most common-reported causes of primary amenorrhea. Gonadal failure or hypergonadotropic hypogonadism is a condition of ovaries not working well despite sufficient gonadotropin stimulation. The condition can either be related to chromosomal abnormalities, autoimmune diseases or iatrogenic. Hypogonadotropic hypogonadism or central hypogonadism is caused by the failure of the hypothalamus or the pituitary to secrete sufficient gonadotropin-releasing hormone (GnRH) or gonadotropins. Mullerian agenesis on the other hand is a structural defect of congenital nature where the uterus and upper vagina are normal and well-developed in the presence of normal ovarian functions and secondary sex characteristics [3].

But the etiological spectrum of primary amenorrhea among adolescents is not the same as it is among adults. The physiological ones process of puberty can continue to be unstable during the adolescent period, and therefore, it is difficult to draw the boundary between normal pubertal delay and pathological Structural delayed puberty or constitutional delay of growth and puberty is a benign condition that is common in adolescents and may resemble pathological hypogonadism [4]. This transitory delay should not be confused with the permanent types of hypogonadism, and in these cases, it is necessary to conduct clinical examination, history formation, and hormonal and radiologic research. Lack of proper diagnosis of the underlying cause can result in unwarranted interventions or untimely treatment.

However, another factor that should be taken into consideration in the case of adolescents with primary amenorrhea is the existence of chronic systemic diseases. The hypothalamic-pituitary-ovarian axis may be disrupted by chronic diseases like endocrine diseases, nutritional deficiencies, long-term systemic diseases. The conditions can cause hormonal disregulation, interfere with growth and pubertal development, and lead to reproductive malfunction. Thus, a complex clinical management is needed to assess not just the reproductive anatomy and hormonal status of the adolescent patient but the general health of the patient as well [5].

Primary amenorrhea assessment is usually done in a systematic and step wise procedure. Primary examination is performed with a history of detailed medical history, physical examination, and secondary sexual characteristics. Special emphasis is placed on the growth trends, pubertal development, and the presence or absence of any indicators of endocrine or systemic conditions [6]. Hormonal tests, including follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin, thyroid function tests, and other endocrine parameters of interest, may be included in the laboratory tests. The presence and the structure of reproductive organs are frequently evaluated by imaging methods, such as pelvic ultrasonography and magnetic resonance imaging (MRI). Genetic studies and karyotype analysis can be required in certain cases in order to detect chromosomal abnormalities.

The significance of systematic assessment consists in the fact that there are numerous etiological factors related to primary amenorrhea. Proper diagnosis of the underlying cause does not only enable proper treatment but also gives significant prognostic data of future reproductive functioning and health in general [7]. Moreover, patients and their families can be counseled in a timely manner with the help of early diagnosis, which is especially critical in adolescents who might undergo considerable psychological distress because of delayed puberty and no menstruation [8].

Since the etiology of primary amenorrhea in adolescents is complex and variable, clinicians need to know the etiological spectrum of the condition. The epidemiological patterns might not be similar among the various populations and healthcare environment, and therefore local data could help in enhancing the diagnostic approach and management guidelines. The research on the spread of etiologies in adolescents may also be used to establish the most prevalent underlying conditions and the possible areas in which additional research or clinical awareness should be provided.

The present paper provides the etiologic information of patients who were followed up due to primary amenorrhea. The study will help to define the extent of knowledge about the etiology spectrum of primary amenorrhea in adolescents and highlight the role of the systematic assessment of the patient in forming a proper diagnosis and ensuring effective treatment.

Methodology

Study Design: This study was conducted as a hospital-based observational cross-sectional study aimed at evaluating the etiological spectrum and systematic clinical, hormonal, and radiological evaluation of adolescents presenting with primary amenorrhea. The design was chosen to assess the various causes of primary amenorrhea and to analyze the clinical characteristics and diagnostic findings among affected adolescents presenting to a tertiary care hospital.

Study Area: The study was conducted in the Department of Obstetrics and Gynaecology at Nalanda Medical College and Hospital, Patna, Bihar, India.

Study Duration: The study was carried out over a period of nine months from January 2025 to September 2025

Sample Size: A total of 27 adolescents diagnosed with primary amenorrhea were included in the study. The sample consisted of all eligible cases presenting to the Department of Obstetrics and Gynaecology during the study period who met the inclusion criteria and consented to participate in the research.

Study Population: The study population comprised adolescent girls presenting with primary amenorrhea to the gynecology outpatient department or inpatient services. Primary amenorrhea was defined as the absence of menarche by 16 years of age despite normal development of secondary sexual characteristics, or absence of menarche by 14 years of age in the absence of secondary sexual characteristics. Adolescents fulfilling this definition were systematically evaluated to determine the underlying cause.

Data Collection: Data were collected using a structured case record form that included demographic details, medical history, and clinical examination findings. A detailed history was taken regarding age, family history of delayed puberty or menstrual disorders, nutritional status, chronic illnesses, and developmental milestones. Physical examination included general examination and assessment of secondary sexual characteristics using Tanner staging. Anthropometric measurements such as height and weight were recorded, and body mass index (BMI) was calculated using the formula weight in kilograms divided by the square of height in meters.

Laboratory investigations included measurement of hormonal parameters such as follicle-stimulating hormone (FSH), luteinizing hormone (LH), estradiol, and prolactin. In cases suspected of polycystic ovarian syndrome, fasting blood glucose and fasting insulin levels were measured to assess insulin resistance. The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) was calculated using the formula fasting glucose multiplied by fasting insulin divided by 405, and values greater than 3.82 were considered indicative of insulin resistance. Radiological evaluation with pelvic ultrasonography was performed to assess the presence, size, and morphology of the uterus and ovaries and to identify structural abnormalities of the reproductive tract. Based on hormonal levels, cases were categorized into normogonadotropic, hypergonadotropic, and hypogonadotropic hypogonadism groups.

Inclusion Criteria

Participants were included in the study if they met the following criteria:

- Adolescent girls presenting with primary amenorrhea.
- Age 14–18 years attending the gynecology department.
- Willingness to participate in the study and provide informed consent (with guardian consent where applicable).

Exclusion Criteria

Participants were excluded if they had:

- Secondary amenorrhea.
- History of previous hormonal therapy for menstrual induction.
- Patients with severe systemic illness or incomplete clinical data.
- Patients unwilling to participate in the study.

Procedure: All adolescents presenting with primary amenorrhea during the study period underwent a systematic evaluation. After registration in the gynecology outpatient department or admission to the ward, detailed clinical history was obtained and a thorough physical examination was performed, including assessment of secondary sexual characteristics. Anthropometric measurements were recorded, and necessary laboratory investigations were carried out to evaluate hormonal status. Pelvic ultrasonography was performed to assess internal genital structures. Additional investigations such as karyotyping or advanced imaging were conducted when clinically indicated. Based on clinical findings, laboratory results, and imaging studies, the underlying etiology of primary amenorrhea was identified and classified.

Statistical Analysis: Statistical analysis was performed using SPSS version 25.0. Descriptive statistics including mean and standard deviation were used to summarize continuous variables, while frequencies and percentages were used for categorical variables. The normality of the data distribution was assessed using the Shapiro–Wilk test. As the data were not normally distributed, differences between independent groups were evaluated using the Kruskal–Wallis test. A p-value of less than 0.05 was considered statistically significant for all analyses."

Result

Table 1 shows the diagnostic distribution of patients in the study (N = 29). The most common diagnosis was normogonadotropic hypogonadism, accounting for 14 cases (48.3%). Within this group, the underlying causes included chronic disease (4 cases), insulin resistance/PCOS (4 cases), prolactinoma (3 cases), and Müllerian agenesis (3 cases). Hypergonadotropic hypogonadism was observed in 9 patients (31.0%), with causes such as idiopathic primary ovarian failure (3 cases), 46, XY disorder of sex development (2 cases), Turner syndrome (2 cases), drug-related ovarian failure (1 case), and autoimmune oophoritis (1 case). The least common category was hypogonadotropic hypogonadism, seen in

6 cases (20.7%), including normosmic hypogonadism (3 cases), constitutional delay of growth and puberty (2 cases), and panhypopituitarism (1 case). Overall, normogonadotropic hypogonadism was the predominant diagnosis among the patients.

Table 1: Diagnostic Distribution of Patients (N = 29)	
Diagnosis	**Number (%)**
Normogonadotropic hypogonadism	**14 (48.3%)**
Chronic disease	4
Insulin resistance / PCOS	4
Prolactinoma	3
Müllerian agenesis	3
Hypergonadotropic hypogonadism	**9 (31.0%)**
Idiopathic primary ovarian failure	3
46,XY disorder of sex development	2
Turner syndrome	2
Drug related ovarian failure	1
Autoimmune oophoritis	1
Hypogonadotropic hypogonadism	**6 (20.7%)**
Normosmic hypogonadism	3
Constitutional delay of growth and puberty	2
Panhypopituitarism	1

Table 2 presents the auxological and laboratory data of patients according to the type of hypogonadism (N = 29). The mean age of patients was comparable among the groups, with 15.31 ± 1.44 years in normogonadotropic, 16.02 ± 1.51 years in hypergonadotropic, and 15.48 ± 1.36 years in hypogonadotropic hypogonadism, showing no significant difference ($p = 0.472$). Similarly, parameters such as height, height SDS, weight, weight SDS, BMI, and BMI SDS did not differ significantly between groups ($p > 0.05$). However, significant differences were observed in hormonal parameters. FSH and LH levels were markedly elevated in hypergonadotropic hypogonadism (86.74 ± 7.12 IU/mL and 33.12 ± 2.71 IU/mL, respectively), while lower levels were seen in hypogonadotropic hypogonadism. Estradiol levels were highest in normogonadotropic hypogonadism (58.64 ± 7.92 pg/mL) and significantly lower in the other two groups ($p = 0.002$). These findings highlight significant hormonal variations among the different types of hypogonadism despite similar auxological characteristics.

Table 2: Auxological and Laboratory Data of Patients According to Diagnosis (N = 29)				
Parameter	**Normogonadotropic hypogonadism Mean ± SD (n=14)**	**Hypergonadotropic hypogonadism Mean ± SD (n=9)**	**Hypogonadotropic hypogonadism Mean ± SD (n=6)**	**p-value**
Age (years)	15.31 ± 1.44	16.02 ± 1.51	15.48 ± 1.36	0.472
Height (cm)	151.9 ± 7.1	152.6 ± 6.8	153.1 ± 6.5	0.881
Height SDS	−1.41 ± 1.02	−1.22 ± 1.61	−1.30 ± 1.42	0.964
Weight (kg)	48.8 ± 13.9	47.9 ± 14.5	45.2 ± 12.6	0.623
Weight SDS	−0.71 ± 1.56	−0.94 ± 1.60	−1.52 ± 1.95	0.417
BMI (kg/m²)	21.9 ± 5.1	20.3 ± 4.3	18.7 ± 2.8	0.116
BMI SDS	0.04 ± 1.38	−0.03 ± 1.01	−0.39 ± 1.05	0.356
FSH (IU/mL)	4.12 ± 1.76[a]	86.74 ± 7.12[a]	1.02 ± 0.98[a]	0
LH (IU/mL)	4.41 ± 3.58[a]	33.12 ± 2.71[a]	0.18 ± 0.29[a]	0
Estradiol (pg/mL)	58.64 ± 7.92[a]	10.31 ± 1.28[a]	8.74 ± 1.11[a]	0.002

Discussion

The most common type in the current study was normogonadotropic hypogonadotropic (48.3, 31.0, and 20.7% respectively). The findings demonstrate partial similarity with already published research where normogonadotropic causes were also significant percentage of cases. As an example, a series of 295 primary amenorrhea patients who received retrospective follow-ups on the same indicated that approximately 50 percent of the patients had normogonadotropic hypogonadism, and about one-third of patients had hypergonadotropic hypogonadism (Tanmahasamut et al., 2012) [9]. Likewise, Rattanachayyanont et al., (1997) [10] have also found that the most frequent causes in cohort of Taiwanese patients with PA were Müllerian agenesis, gonadal dysgenesis and hypothalamic hypogonadism. Unlike such reports, the current study showed relatively low rates of Mullerian agenesis (10.3%), which can

be explained by the previous clinical diagnoses of other endocrine etiologies or different referral practices."

Normogonadotropic hypogonadotropism was almost fifty percent of the cases of the current series. Among the most common etiologies in this group, there were chronic diseases, insulin resistance linked to polycystic ovary syndrome (PCOS). Past findings have likewise indicated that systemic illnesses and metabolic disturbances are the factors that are disruptive to the hypothalamic-pituitary-ovarian axis in causing the menstrual abnormalities. Pulsatile secretion of gonadotropin-releasing hormone (GnRH) and luteinizing hormone (LH) can be impaired by chronic diseases and, thus, lead to disrupted pubertal development and menstruation (Meczekalski et al., 2008) [11]. Chronic disease was found in 13.8% of patients in our study which is similar to previous findings that showed a range of 10-15 per cent of adolescents with primary amenorrhea may have underlying systemic pathology affecting the reproductive functioning (Sharma et al., 2008) [12].

The present study found PCOS and insulin resistance in 13.8 cases. The literature has been used to describe similar rates where 1.4-14% of adolescents presenting with primary amenorrhea have been reported to have PCOS (Dramusic et al., 1997) [13]. There are studies which have proposed that adolescent girls with PCOS who manifest with primary amenorrhea are likely to have more severe metabolic disruptions than those who manifest with secondary amenorrhea or oligomenorrhea (Rachmiel et al., 2008) [14]. This is in line with the fact that our study has reported a relative higher mean BMI of normogonadotropic group (21.9 ± 5.1 kg/m 2) as opposed to the other groups even though the difference between the groups is not significant. The rise in morbidity of obesity and metabolic syndrome in teenagers could lead to the rising awareness of PCOS as a relevant etiology of postponed menarche and menstrual abnormalities.

Another significant endocrine etiological factor of primary amenorrhea is hyperprolactinemia as an increased level of prolactin suppresses hypothalamic GnRH release and disrupts the reproductive axis. The current research found a prevalence rate of prolactinoma at 10.3 in the normogonadotropic group. This percentage is equivalent to previous pediatric endocrine series that have documented hyperprolactinemia in about 8 -12 percent of the teenage with menstrual disturbances (Eren et al., 2011) [15]. Prolactinoma has to be identified early since the administration of dopamine agonists usually restores normal ovulation and fertility.

In our cohort, hypergonadotropic hypogonadotrophic was found in 31.0%. The most common etiology in this category was idiopathic primary ovarian failure (POF), then chromosomal abnormalities (Turner syndrome and 46, XY disorders of sex development). Gonadal dysgenesis and primary ovarian failure have been reported by previous literature as the most common causes of hypergonadotropic hypogonadism in secondary amenorrhea in adolescents (Goswami and Conway, 2007) [16]. Genetic factors are gaining importance in pathogenesis of ovarian insufficiency and some of these genetic defects are identified as BMP15 and POF1 / POF2 locus as causative of ovarian failure (Di Pasquale et al., 2004) [17]. In our research idiopathic ovarian failure was the cause of about one-third hypergonadotropic cases which is congruent to previous studies that most patients are left without a distinctly definable etiology despite undergoing thorough examination.

Another cause of primary amenorrhea that is well-known is chromosomal abnormalities. Past reports include the chromosomal abnormalities in 15.963.3% of patients with PA, of which the most frequent was the Turner syndrome (Mondal et al., 2002) [18]. In the current research, chromosome disorders were seen in 13.8 per cent of cases involving Turner syndrome and 46, XY disorders of sex development. This percentage is a little bit lower than the ones which are reported in some previous studies, but this fact can be attributed to the fact that the sample size is small and there is a regional difference in patient characteristics.

The present series had hypogonadotropic hypogonadism as 20.7 percent. Normosmic hypogonadism and constitutional delay of puberty and growth were the most frequently found causes. Hypogonadotropic hypogonadism is caused by a lack of release or an absence of response to secretion of GnRH and can be caused by birth defects, genetic mutations, or by functional suppression of the hypothalamus (Bhangoo & Jacobson-Dickman, 2009) [19]. Gene mutations have been identified to cause normosmic hypogonadotropic hypogonadism, and these include TAC3, GPR54, FGFR1, and PROK2 but genetic testing was not conducted in the current study (Meczekalski et al., 2008) [11]. The occurrence of constitutional delay among subsets of patients underscores the need to exercise caution when making a clinical assessment and follow-up studies in order to make a definite diagnosis.

The parameters of auxology in our study were generally similar within the diagnostic groups and there was no statistically significant difference in age, height, weight, and BMI. Such an observation implies that anthropometric measurements might not be the reliable means of differentiating various etiologies of primary amenorrhea. The same has been reported in earlier studies where it was found that clinical and growth parameters were much overlapping across diagnostic categories (Timmreck & Reindollar, 2003) [20]. Conversely, the hormonal parameters exhibited significant differences among

groups, which is a representation of the pathophysiological processes. The high levels of FSH and LH in the hypergonadotropic hypogonadotropic observation in the present study and the very low levels of the hormones in hypogonadotropic hypogonadism are in line with the known endocrine patterns in literature (Welt, 2008) [21].

Altogether, the results of the current research indicate the heterogeneity of etiological range of primary amenorrhea among adolescents. The most frequent etiologies in our cohort were normogonadotropic etiologies, especially metabolic and endocrine etiologies: PCOS and hyperprolactinemia, but the gonadal failure and hypothalamic pituitary disorders had smaller, yet clinically important proportions. These results highlight the significance of a gradual clinical, hormonal and cytogenetic assessment to diagnose and treat adolescents with primary amenorrhea efficiently and promptly.

Conclusion

The present study highlights the diverse etiological spectrum of primary amenorrhea in adolescents and underscores the importance of a systematic clinical and laboratory evaluation for accurate diagnosis. Normogonadotropic hypogonadism constituted the most common diagnostic category, with conditions such as chronic systemic illness, insulin resistance/PCOS, prolactinoma, and Müllerian agenesis being notable contributors. Hypergonadotropic hypogonadism formed the second most frequent group, including causes such as primary ovarian failure, disorders of sex development, Turner syndrome, and autoimmune or drug-related ovarian damage. Hypogonadotropic hypogonadism accounted for a smaller but significant proportion, mainly due to normosmic hypogonadism, constitutional delay of growth and puberty, and pituitary disorders. While auxological parameters were largely comparable across diagnostic groups, significant differences in gonadotropin and estradiol profiles helped differentiate the underlying etiologies. Overall, the findings emphasize that a structured approach integrating clinical assessment, hormonal evaluation, and etiological classification is essential for timely diagnosis and appropriate management of adolescents presenting with primary amenorrhea.

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