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PUBERTY MENORRHAGIA IN A RURAL MEDICAL COLLEGE

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Abstract

Background: Puberty menorrhagia can pose a significant challenge to the gynecologist when associated with serious systemic complications like anaemia and hypoproteinaemia. Early diagnosis and treatment with individualization of each and every case is the keystone in the management of puberty menorrhagia. Exclusion of pregnancy is mandatory in every case, irrespective of the history, reassurance, counseling, regular follow-up along with a balanced nutritional diet and long term iron therapy go a long way in successful management of such cases.

Setting: This study was conducted at PRM Medical College, Baripada, Odisha, India from January 2018- December 2020.

Methods: Data was collected from medical case records in each of these cases from indoor case sheets and from the patients attending the gynaecology OPD.

Results: There were 70 indoor admissions in the gynaecology l puberty menorrhagia over a span of two years. The leading cause was anovulatory dysfunctional uterine bleeding. Other systemic associations included hypothyroidism, idiopathic thrombocytopenic purpura, genital tuberculosis, and PCOD. Each case was analyzed for demographic profile, duration of menorrhagia, severity of symptoms, degree of anaemia, final diagnosis, requirement of blood and component therapy and response to conservative management.

Conclusions: Most abnormal bleeding in adolescents is caused by immaturity of the hypothalamic - pituitary ovarian axis resulting in anovulation. Approximately 20% of adolescents have an underlying endocrine or haematological disorder requiring targeted diagnostic testing.

Keywords: Puberty menorrhagia, Dysfunctional uterine bleeding, Hormonal therapy

INTRODUCTION

Menarche is the transition from childhood to puberty. Although mechanisms triggering puberty and menarche remain uncertain, they are dependent on genetics, nutrition, body weight and maturation of the hypothalamic pituitary- ovarian axis. The complete maturation of the axis may take up to 2 years. During this time, it is common for adolescents to present with complaints of menstrual irregularities. Abnormal bleeding accounts for approximately 50% of gynaecological visits in adolescent girls.1 These complaints encompass disorders ranging from minimal spotting to profuse

bleeding. Puberty menorrhagia is defined as excessive bleeding occurring between menarche and 19 years. In 80% of cases puberty menorrhagia is caused by anovulatory cycles.3 There is an immaturity of the hypothalamus and inadequate positive feedback resulting in sustained high levels of estrogen. An organic disease or malignancy in particular is very rare. In all cases of puberty menorrhagia it is mandatory to exclude pregnancy, especially an incomplete abortion or ectopic pregnancy. In persistent abnormal bleeding coagulation disorders and leukemia should be ruled out. Occasionally menorrhagia is the only presenting symptom in a patient of coagulation disorders.2 In general the prognosis is better when dysfunctional uterine bleeding starts after a period of regular menstruation than when it starts at menarche.4 The main causes of abnormal uterine bleeding adolescent are Uterine causes as Pregnancy, endometritis, hyperplasia, malignancy, polyp, Leiomyomata ,Ovarian causes asImmature hypothalamic pituitary ovarian axis, pcod, estrogen producing tumors ,Cervical causes as Cervicitis, condyloma, sarcoma botryoides, polyp, malignancy ,Vulval/vaginal causes asTrauma, vaginitis, infection, sarcoma botryoides Endocrine causes as Hypothyroidism, hyperprolactinaemia and Coagulation disorders . during puberty, maturation of the hypothalamic pituitary - ovarian axis is characterized by an increase in the frequency and amplitude of pulsatile GnRH, which initiates and regulates secretion of pituitary gonadotropins.9

During the prepubertal years, LH is secreted primarily at night in an episodic fashion. With the progression to puberty, LH peaks increase in a pattern similar to that seen at night. The timing of these LH pulses is crucial in establishing normal ovulatory cycles. Increases in basal LH as well as immature timing of pulses result in anovulatory cycles. These cycles are characterized by levels of LH and FSH secretion that are sufficient to induce follicular development and estrogen production but inadequate to induce follicular maturation and ovulation. Thus unopposed estrogen stimulates endometrial growth. This ultimately outgrows its blood supply and architectural support, resulting in partial breakdown and shedding in an irregular manner.In the proliferative phase the endometrium synthesizes equal amounts of PGE2α (Vasoconstrictor and weak platelet aggregator) and PGE2 (Vasodilator with weak platelet antiaggregatory effect). However in the luteal phase the levels of PGE2α progressively increase under the influence of estradiol and progesterone. In normal menstruation the ratio of PGE2α:PGE2 is 2:1 so that it is the vasoconstrictor and platelet aggregator action that predominates. In anovulatory DUB the lack of progesterone results in decrease in the PGE2α:PGE2 ratio and relative increase in the vasodilator and antiplatelet aggregatory PGE2 which would account for the increased mean menstrual blood loss. It could also account for the absence of uterine contractions and painless periods characteristics of anovulatory menstruation.

The most common coagulation disorders were idiopathic thrombocytopenic purpura, Von Willebrands disease, and leukemia and platelet dysfunction like Glanzmanns thromboasthenia. Young girls with blood coagulopathies are at a high risk for abnormal bleeding with the onset of menarche,9 and must be treated appropriately at the time of puberty. Laboratory evaluation, including a complete blood count, platelets, prothrombin time, partial thromboplastin time and bleeding time provides an adequate screen for coagulation disorders. Hypothyroidism can be associated with pubertal DUB. The reported incidence of subjective menorrhagia in myxoedema varies from 32-80% and menorrhagia may not infrequently be the presenting complaint (Scoot and Massey 1964). The menorrhagia associated with hypothyroidism responds promptly to the thyroid replacements, often in doses insufficient to correct the other manifestations of the condition. This suggests that thyroxine does have a direct effect on the spiral arterioles and on haemostasis at menstruation.4

The goals of treatment in adolescents are to regulate menstruation and decrease hirsuitism and acne. The best treatment modality is an oral contraceptive pill because of the inhibition of LH and decrease in circulating testosterone levels. Sex hormone -binding globulin is increased and available to bind and inactivate testosterone in the circulation.7 Oral contraceptive pills do not seem to aggravate the underlying insulin resistance significantly and may attenuate some of the lipid derangement’s induced by sustained excess androgen exposure8 Another treatment option to control abnormal bleeding is cyclic medroxyprogesterone acetate 10 mg orally for 10 days of each month.

However this regimen does not alleviate the associated androgenic effects of polycystic ovaries.9 To summarize, Blood and component therapy along with hormones constitute the main medical therapy in the treatment of critical puberty menorrhagia. In the present study 6 patients required intravenous conjugated estrogen. Various studies suggest that these are of value in arresting profuse haemorrhage4 and are usually given with antiemetics. Oral contraceptive pills were administered to 19 patients in our study group. These form an effective hormonal therapy to restore the balance between prostaglandins and thromboxane A2 and reduce the mean menstrual blood loss with additional cycle stabilization. 45.7% patients in our study series had received progestogens for medical curettage. Androgenic progestogens alone may be used to arrest uterine haemorrhage or administered cyclically throughout the menstrual cycle (5th to 25th day) norethisterone acetate (primolut N) 20-30 mg daily is given to arrest haemorrhage and not more than 3 days. The progestogen may then be continued at a lower dose for upto 21 days. The patient should be warned that a withdrawal bleeding will occur on stopping treatment that will cease in 4-5 days. Norethisterone acetate can also be given from 5th to 25th day in a dose of 5 mg once a day. Androgens like danazol are less favoured because of their masculinizing side effects in adolescent girls.

Antifibrinolytic like tranexamic acid are a newer form of treatment in puberty menorrhagia. Plasminogen activators are a group of enzymes that cause fibrinolysis. An increase in the levels of plasminogen activators has been found in the endometrium of patients with heavy menstrual bleeding compared to those with normal menstrual loss. Plasminogen activators have been therefore been prompted as a treatment in heavy menstrual bleeding.3 There has been reluctance to prescribe tranexamic acid due to possible side effects of the drugs such as thrombogenic disease (DVT). Long term studies in Sweden; however have shown that the rate of incidence of thrombosis in women treated with tranexamic acid is comparable with the spontaneous frequency of thrombosis in women.3 Ethamsylate was used in 8 of our patients. It reduces capillary bleeding when the platelets are adquate; probably it corrects abnormal platelet function. It is not an antifibrinolytic. It does not stabilize fibrin. Majority of patients in the study group received a combination therapy with OCPs with androgens or with progesterone or with antifibrinolytic agents.12

MATERIAL AND METHODS

The present ongoing study evaluates 70cases of critical puberty menorrhagia managed at PRM Medical College ,Baripada,over a period of 2 years from January 2018 to December 2020. Data was collected from medical case records in each of these cases from indoor and OPD case sheets. A proforma was made and data entered and analyzed from each proforma. An approval was taken from ethical committee of hospital. It was a prospective observational study and no intervention was done. Each case was evaluated for the demographic profile, severity of symptoms, degree of anaemia, final diagnosis, requirement of blood and component therapy and response to conservative management. The baseline investigations in all the cases included exclusion of pregnancy by urine testing, complete blood count, peripheral smear for RBC and WBC morphology, coagulation profile, blood grouping and Rh typing and transabdominal USG. In selected cases thyroid function test (T3, T4, TSH) and hormonal assays including (LH, FSH, Prolactin) and chest X-ray were done.

OBSERVATION

Our study showed that 50% of patients were in the age group of 13-15 years. Majority of the patients belonged to the lower socioeconomic status. Almost 60% of patients had onset of menorrhagia since less than 6 months. Of these 33% of patients had haemoglobin less than 6 Gm%. Anovulatory dysfunctional uterine bleeding occurred in 56 cases (80%). Four patients of secondary DUB had a final diagnosis of idiopathic thrombocytopenic purpura, Four patients were diagnosed to have hypothyroidism. Genital tuberculosis was detected in another 4 cases. Analysis of data showed that 37% of patients received fresh blood transfusion, 8% required component therapy in the form of fresh frozen plasma, platelets and cryoprecipitates. Majority of patients received a combination medical therapy . Majority of patients received OC pills + ethamsylate (20%). Six

patients received i.v. oestrogens plus progesterone while ethamsylate along with progestogens was used in 5 patients. Tranexamic acid was used with biphasic OC pills in 17.1% of cases and with progestogens in 8.5% cases.

TABLE 1:AGE DISTRIBUTION

Age group	Number of cases	Percentage
13-15	35	50
16-17	18	25.72
18-19	17	24.28

TABLE 2: DURATION

Duration of symptoms	Number	Percentage
Less than 6 months	42	60
6months to 1 year	14	20
More than 1 year	14	20

TABLE 3: CAUSES OF PUBERTY MENORRHAGIA

Causes	Number	Percentage
Anovulatory uterine bleeding	56	80
ITP	4	5.71
Hypothyroidism	4	5.71
Genital Tb	2	2.85
PCOD	2	2.85
Vaginitis	2	2.85

TABLE 4: TREATMENT REGIMES

Therapy regime	Number	Percentage
Biphasic OCpills +ethamsylate	14	20
Biphasic OCpills + tranexamic acid	12	17.1
IV estogen + progesterone	12	17.1
OCP + testosterone in acute phase	12	17.1
Progestogen + ethamsylate	10	14.28
Progestogen + tranexamic acid	6	8.57
Progesterone	4	5.71

CONCLUSION

In conclusion, most abnormal bleeding in adolescents is caused by immaturity of the hypothalamic - pituitary ovarian axis resulting in anovulation. Approximately 20% of adolescents have an underlying endocrine or haematological disorder requiring targeted diagnostic testing. Individualizing every case, excluding pregnancy, timely hospitalization, a thorough history, physical examination and base line workup are crucial in the management of every case. Reassurance, counseling of adolescent girls about reproductive physiology, regular follow-up, balanced diet and long term iron therapy go a long way in treatment of puberty menorrhagia.

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