ORIGINAL ARTICLE J. Med. Sci. (Peshawar, Print) January 2012, Vol. 20, No. 1: 15-18

PUBERTY MENORRHAGIA: CAUSES AND MANAGEMENT

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ABSTRACT

Objectives: To determine the causes of Puberty Menorrhagia and to evaluate the efficacy of Medical Management.

Material and Methods: This descriptive study included 35 patients, who presented with puberty menorrhagia to Gynae C Unit Khyber Teacing Hospital, Peshawar from January 2008 to December 2010. Assessment of each case with thorough history, physical examination and laboratory investigations was done.

Results: In 26 (74.2%) patients immaturity of hypothalamic pituitary ovarian axis was the cause of puberty menorrhagia. three (8.6%) patients had polycystic ovarian disease (PCOD), 1 (2.8%) patient had hypothyroidism, 3 (8.6%) had thrombocytopenia, 1 (28%) patient had VonWillebrand disease (vWD), 1 (2.8%) patient had multiple fibroids in the uterus. Twenty (57.14%) patients had menorrhagia of > 1years duration. Six (17.11%) patients had haemoglobin level of < 5gm/dl. One (2.8%) patient needed surgical intervention in the form of myomectomy. All other patients responded to medical management.

Conclusion: Puberty menorrhagia is a distressing condition both for the patient as well as her parents. Most of the cases are due to anovulatory dysfunctional uterine bleeding (DUB) which is a self limiting condition. Counselling of the patients is an important part of management. Long term medical treatment is successful in the majority of cases.

Key Words: Puberty, Menorrhagia, Causes, Management.

INTRODUCTION

Puberty is defined as the state of being functionally capable of procreation. The term is generally used in a more comprehensive sense to refer to the whole period of time during which secondary sexual characteristics develop, menstruation begins in females and psychosexual outlook of a human being changes. There are five main physical features of puberty: breast growth, pubic hair growth, axillary hair growth, increase in height and menstruation. The onset of menstruation is influenced by a number of factors: genetics, nutrition, body weight and maturation of the hypothalamic pituitary ovarian axis. The onset of menstruation does not mean that ovulation has occurred; in the majority early menstrual cycles are anovulatory. The cycle length varies for some considerable years after menarche. It may take some 5-8 years before menstrual cycle normality is established. During this time it is common for adolescents to present with menstrual irregularities[1].

Young girls with blood coagulopathy are at a high risk of abnormal bleeding with the onset of menarche. Bleeding is usually heavy, causing anaemia and may require blood transfusion. Among the inherited bleeding disorders platelet defects are the most common causes of puberty menorrhagia[2]. Adolescents with gynaecological problems require a degree of privacy and sensitive handling, as many of the gynaecological problems encountered relate to intimate body functions at a time when the individual is maturing sexually and having to deal with issues that are embarrassing and may be considered taboo. This study was conducted to find out the causes of puberty menorrhagia in our setup, and role of conservative management.

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MATERIAL AND METHODS

A total of 35 young girls from age of menarche to 19 years with history of excessive bleeding per vagina attending out patient department or admitted to the Gynae C Unit, Khyber Teaching Hospital, Peshawar were included in the study. The study was carried out from January 2008 to December 2010. Blood loss during menstruation was considered excessive if the duration of menstruation was more than seven days and/or there was a history of passage of clots and the haemoglobin level was < 10gm/dl. A detailed history regarding age of patient, age of menarche, previous menstrual history was taken. The presenting complaints about onset, duration and amount of blood loss were noted. Requirement of blood, component therapy and response to previous therapy were also recorded. The medical history included history of recent weight change, tuberculosis, thyroid disorders and haema-tological disorders. Past surgical history of any excessive bleeding was noted. Personal history included history of any drug intake. Family history was taken in detail

regarding presence of any disease like tuberculosis, thyroid disease and bleeding diathesis.

Patients were examined for pallor, lymphadenopathy and gum bleeding. The pulse and blood pressure were recorded. Abdominal palpation was done for hepatosplenomegaly and abdominal masses. Tenderness in sternum and other bony areas and joint swelling was looked for. Skin was examined for any purpuric spots, acne, hirsutism and features of hyperandrogenism. A protocol of investigation was made. The base line investigation were done including Pelvic ultrasound for uterine and ovarian morphology.

Some investigations (VonWillebrand factor activity, Ristocetin cofactor assay, 21 day serum progesterone level) were done in selected patients. The management protocol depended upon the condition of the patient and the underlying cause of menorrhagia. In anovulatory bleeding with a hemodynamically stable patient, prostaglandin synthetase inhibitors like mefenamic acid and antifibrinolytic drugs like tranexamic acid were used as first line therapy during the days of menstruation for control of blood loss. Hormones like oral contraceptive pills, progestins were prescribed in cases not responding to non-hormonal therapy. Anaemia was corrected by oral hematinics or blood transfusion / component therapy in consultation with a haematologist. Specific treatment for correction of hematological disease and thyroid disease was carried out.

RESULTS

In this study most of the patients were of 12-13 years (37%) and only 45% were above 13 years. The symptom with puberty menorrhagia remained upto one year in 57.14% of cases as shown in Table 1. Different causes of anaemia and hemoglobin levels are shown in Table 2. In 74.28% the cause of puberty menorrhagia was anovulatory dysfunctional uterine bleeding, the other causes are shown in Table 3.

Thirteen (37.17%) patients responded well to iron and a 3-5 day course of tranexamic acid in the dose of 1-2 gm/day. Fifteen (42.86%) patients responded to oral progesterone for 3 cycles. Five (14.3%) patients responded to oral contraceptive pills. The hypothyroid patient responded to thyroxine therapy. Patients with decreased platelet count were initially treated with blood and platelet transfusion, prednisolone therapy and oral tranexamic acid and were put on medroxyprogesterone acetate from day 5-25 of the cycle. The patient with Von Willebrand disease responded well to oral progesterone and desmopressin nasal spray.

Table 1: Duration of Symptoms

Duration	No. of patients & percentage
Less than 06 months	4 (11.42)
06 months – 1 year	11 (31.42)
more than 1 year	20 (57.14)

Table 2: Haemoglobin Percentage of Patients

Causes	Haemoglobin level in gm /dl		
	<5 gm /dl	5.1 –7 gm/dl	7.1–10 gm/dl
	No. of patients & %age		
Von Willebrand disease	1(2.86)		
Dysfunctional uterine bleeding	1 (2.86)	8 (22.85%)	17(48.57)
Fibroid uterus	1(2.86)		
Thrombocyto-penic purpura	3(8.57)		
Polycystic ovarian disease		2(5.71%)	1(2.86)
Hypothyroidism		1(2.86%)	

Table 3: Etiology of Puberty Menorrhagia

Causes	No. of patients and %age
Anovulatory dysfunctional Uterine bleeding	26 (74.28)
Polycystic ovarian disease	3 (8.6)
Hypothyroidism	1 (2.8)
Thrombocytopenic purpura	3 (8.6)
Von Willebrand disease	1 (2.8)
Fibroid Uterus	1 (2.8)

DISCUSSION

Menarche is a hallmark event in the life of adolescent girls, it marks the transformation from childhood to puberty. Puberty menorrhagia is defined as excessive bleeding in amount > 80ml or in duration > 7 days between menarche and 19 years of age. During puberty, maturation of the hypothalamic pituitary ovarian axis is characterized by an increase

in frequency and amplitude of pulsatile GnRH, which initiates and regulates secretion of pituitary gonadotrophins[3,4]. During prepubertal years LH is secreted primarily at night in an episodic fashion. With the progression to puberty, day LH peak increases in a pattern similar to that seen at night. The timing of these LH pulses is crucial in establishing normal ovulatory cycles. Increase in basal LH as well as immature timing of pulses results in anovulatory cycles. These cycles are characterized by levels of LH and FSH secretion that are sufficient to induce follicular development and oestrogen production but inadequate to induce follicular maturation and ovulation. Thus unopposed oestrogen stimulates endometrial growth. This ultimately outgrows its blood supply and architectural support, resulting in partial breakdown and shedding in an irregular manner[1].

In proliferative phase, the endometrium synthesizes equal amounts of PGF2 (vasoconstrictor and weak platelet aggregator) and PGE2 (vasodilator and weak platelet antiaggregator). However in the luteal phase the level of PGF2 progressively increases under the influence of oestrogen and progesterone. In normal menstruation, the ratio of PGF2: 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 decreased PGF2: PGE2 ratio and relative increase in vasodilator PGE2 which could account for increased mean menstrual blood loss. It could also account for absence of uterine contractions. This can be a recurrent problem until the cycle becomes regular. Occasionally anemia results with a haemoglobin level as low as 6 or 7 gm/dl[5].

In our study 74.28% of cases of puberty menorrhagia were due to anovulatory dysfunctional uterine bleeding. Chaudury et al reported 71%[6], Roychowdhury 61.5%[7], Neinstein 95%[8] of cases of puberty menorrhagia as being due to anovulation due to immaturity of hypothalamic pituitary ovarian axis. Most of these patients had already received a combination of medical regimes which had improved their condition. However on stopping the medicines their bleeding recurred. Treatment is directed towards stabilizing the endometrium and treating the hormonal alterations. It includes reassurance that this is a self limiting problem. First line treatment in mild cases is tranexamic acid and NSAIDS during the menstrual cycle[9]. Tranexamic acid is effective, safe, the bioavailability is 35% which requires administration of at least 1 gm 4-6 hourly[10]. Hormonal treatment is required where the girl is anaemic or where the problem is recurrent and restricts her activity for 3-6 months.

Progesterones alone are generally effective but can be used in combination with estrogen. Progesterone can be used cyclically in two different treatment protocols: as a short course during the luteal phase and a relatively longer course lasting 21 days from day 5 of the cycle. Heavy bleeding can be treated with (1) oral Medroxyprogesterone 10 mg three times /day for 14 days. (2) Medroxyprogesterone acetate injection (Depo Provera) 150 mg intramuscularly every 12 weeks. (3) Progesterone can also be used for medical curettage, in the form of norethisterone acetate 20-30 mg daily for 3 days to arrest haemorrhage. It may then be continued at a lower dose for up to 21 days. Withdrawal bleeding will occur on stopping the treatment that ceases in 4-5 days. (4) Oral contraceptive pills taper using monophasic pills can also be given, 4 pills evenly spaced per day for 4 days, 3 pills per day for 3 days, 2 pills per day for 2 days and 1 pill /per day for 2 months without taking the placebo pill[11].

In patients with severe bleeding associated with haemodynamic changes blood transfusions are indicated with administration of intravenous conjugated equine oestrogen, 25 mg I/V every 4 hours for upto 24 hours. Bleeding usually decreases within 24 hours and then oral oestrogen can be substituted. A progesterone is also usually added[12]. In Polycystic ovarian disease, ovaries typically have multiple follicular cysts less than 10mm in size and increased stroma. It is associated with chronic anovulation and hyper androgenism. The primary menstrual irregularity in these patients is oligomenorrhoea, although about 5% may demonstrate polymenorrhoea. This may be temporary in adolescents or may eventually progress to advanced polycystic ovarian disease. Diagnosis is confirmed with altered cycle day 2 luteinizing hormone and follicle stimulating hormone ratio. These patients are prescribed hormonal pills.

Young girls with blood coagulopathies are at a high risk of abnormal bleeding with the onset of menarche, bleeding is usually heavy causing anaemia and may require blood transfusion. Claessen et al found 20% of cases of menorrhagia to be due to primary coagulation disorders.[13]. In our study 4(11.4%) patients had coagulation defects. Platelet function defects are an important cause of menorrhagia. Saxena[14] et al found platelet function disorder in 83% of women with menorrhagia due to coagulation defects.

Phillip et al[2] reported an incidence of abnormal platelet aggregation in 45% of women with bleeding disorder. In our study 3 patients (8.6%) had idiopathic thrombocytopenic purpura. Acute idiopathic thrombocytopenic purpura is most commonly seen in the young and is immunological, caused by immune complex containing viral antigens that bind to platelet Fc receptors or by antibodies produced against viral antigens that cross react with platelets. The majority of studies in the west report von Willebrand disease as the most common inherited bleeding disorder leading to menorrhagia whereas studies in South East Asia have found platelet function disorders as the leading inherited bleeding disorder in women with menorrhagia[15].

VonWillebrand factor is a central component of haemostasis serving both as a carrier of factor VIII and as an adhesive between platelets and injured blood vessels[16]. Defects in VWF cause bleeding by impairing both platelet adhesion and blood clotting.

Von Willebrand disease has a prevalence of 0.7-1.6 % in the general population. The high prevalence of menorrhagia among women with Von Willebrand disease has been reported in several studies. In a survey of 99 patients with type 1 Von Willebrand disease from 4 hemophilia centers in USA, 78% reported their periods to be heavy, 71% of whom required medical attention and 15% required hysterectomy[17]. In a study by Rick et al[18] 93% of 38 patients with Von Willebrand disease suffered from menorrhagia. It was also the commonest initial bleeding symptom leading to a diagnosis of the disorder in 53% of them and in all this started from menarche. Although now reports are available on the prevalence of Von Willebrand disease in Pakistan some of the tertiary care centers and institutions have reported a prevalence of 3.18-7.74% among cases with hereditary bleeding disorders[19].

Endocrine disorders can cause anovulation producing an environment of unopposed oestrogen. In the absence of progesterone, the endometrium eventually breaks down causing menorrhagia[20]. The menorrhagia associated with hypothyroidism responds to thyroid replacement therapy in doses sufficient to correct the other manifestations of the condition. The present study showed one patient with fibroid uterus having menorrhagia. She had myomectomy done at 18 years of age, due to excessive bleeding. She developed recurrent fibroids and again presented with severe bleeding. She was given gonodotrophin releasing hormone analogue for 3 months but ultimately needed repeat myomectomy.

CONCLUSION

Abnormal menstrual bleeding in adolescents can be caused by a number of conditions, the most common cause is immaturity of the hypothalamic pituitary ovarian axis. Bleeding disorders are another. Assessment of each case with thorough history, physical examination, and laboratory investigations are crucial in reaching the diagnosis. Once a proper diagnosis is made, counseling of the patient and her parents, follow up and long term therapy in some cases is required.

REFERENCES

1. D Keth Edmonds. Gynecological disorders of childhood and adolescence Dewhursts textbook of obstetrics and gynecology -7TH edition Blackwell Publishing 2007; 364-68.
2. Phillipp CS, Faiz A, Dowling N, Dilley A, Micheals LA et al Age and prevalence of bleeding disorders in women with menorrhagia Obstet gynaecol 2005; 105: 61-68.
3. Delemarre HA, Wennink JM, Odink RJ. Gonadotrophin and growth hormone secretion throughout puberty. Acta Paediatrica Scandinavica 372 (Suppl): 26-31, 1991.
4. Mauras N, Rogol AD, Haymond MW. Sex steroid, growth hormone, insulin lyke growth factor – 1: Neuroendocrine and metabolic regulation in puberty Hormone Research 1996. 45: 74-80.
5. Rao S, Pawar V, Badhwar VR, Medical intervention in puberty menorrhagia. Bombay hospital journal 2004; 466(2) full text available at www.bhj.org/journal/2004-4602-april
6. Chaudhury S, Bhattacharya PK, Sarkar A. Study of adolescence menorrhagia Indian medical journal 2007; 101(5); 161-64.
7. Roychowdhury J, Chaudhuri S, Sarkar A, Kumar B. A study to evaluate the ethiological factors and management of puberty menorrhagia OJHAS VOL 7 Issue 1; (2008 Jan-Mar) Online journal of health and allied sciences.
8. Neinstein LS Menstural problems in adolescents Med Clin North Am 1990; 74: 1181-90.
9. Royal college of obstetrician and gynecologists The initial management of menorrhagia, RCOG evidence based clinical guidelines No. 1 London 1999.
10. Lethaby A, Farquhar C, Cooke I Antifibrinolytic for heavy menstrual bleeding The Cochrane library issue 1-2003.
11. Linda M, Szymanski, Kimberly B Abnormal uterine bleeding, The John Hopkins manual of Gynecology and obstetrics Third edition page 417-428 Lippincote Williams and wilkins.
12. Devore GR, Owens O, Kase N. Use of intravenous premarin in treatment of dysfunctional uterine bleeding, Double blind randomized control study Obstet Gynaecol 1982; 59: 285-93.
13. Claessens CA, Cowell CA Acute adolescent menorrhagia American journal obstet Gynaecol 1981; 139(3); 277-80.
14. Saxena R, Gupta M, Gupta K, Kashyap R, Choudry VP Inherited bleeding disorder in Indian Women with menorrhagia Haemophilia 2003; 9: 193-96.
15. Nazli H, Nusrat hassan, Tasneem farzana, Tahir Sultan Shamsi Adolescent menorrhagia due to platelet disorder JPMP 1/5/52011.
16. Ruggeri ZM, Ware J Von willebrand factor. FASEB J 1993; 7: 308-16.
17. Rodeghiero F, Castamen G, Dini E Epidemiological investigation of the prevalence of Von Willebrand Disease 454-59.
18. Rick ME. Von Willebrand Disease: Clinical presentation and diagnosis of Von Willebrand Disease. (Online Text Book of Medicine) Wellesley, MA,USA 2007.
19. Saeedi L, Razzik F, URehman M, Ahmed H Von willebrand disease J postgrad medical institute 2001; 15: 202-07.
20. Foster PA The reproductive health of women with von willebrand disease Unresponsive to DDAVD: result of an international survey. On behalf of the subcommittee on von willebrand factor of the scientific and standardization committee of ISTH Thromb Haemostat 1995; 74: 784-90.