CASE REPORT

Severe Adolescent Menorrhagia Due to Cervical Fibroid

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Introduction

The initial cycles following menarche are often anovulatory due to delay in positive feedback mechanism of the hypothalamus–pituitary–ovarian axis. The consequent estrogen secretion in such situation results in endometrial proliferation with unstable growth and incomplete shedding which presents as menorrhagia [1, 2]. Amongst the causes of adolescent menorrhagia, dysfunctional uterine bleeding accounts for the vast majority [3]. Having ruled out anovulation and any pregnant status, excessive bleeding during adolescence should raise the doubt of coagulopathy since this accounts for the other large subgroup especially in abnormal uterine bleeding at menarche [4, 5]. The bleeding secondary to blood dyscrasias is usually a heavy flow with regular, cyclic menses [6]. Anovulation and coagulopathy therefore constitute the majority of cases of adolescent menorrhagia (93–95 %).

Thyroid under or over activity, severe renal or hepatic disorders can also present with menorrhagia. The remaining 5–7 % of cases are due to local causes and include lesions, tumors, infections, foreign bodies and lacerations

of the vulva, vagina and cervix [7]. Sexually transmitted diseases should also be borne in mind. Neoplastic causes are highly unlikely in this age group. Though always a dilemma in adolescents a pelvic examination may be life-saving if diagnosis is unclear. The importance of doing a thorough local examination in spite of absent history of sexual activity is highlighted by the following case.

Case Report

A 17 year old girl was referred for tertiary care to our centre at midnight on 8 August 2004 with severe menorrhagia often days duration and severe anemia (Hb 5 g%). She had been managed with low dose OC pill by a consultant gynecologist elsewhere for the preceding 3 days to which she had not responded. One unit of fresh blood had already been transfused and the second unit was on flow on arrival to the hospital. She was bleeding heavily using a pad per hour with passage of clots during each change associated with dull, colicky pain in the lower abdomen. She had been diagnosed as a case of polycystic ovarian disease following menarche at the age of 14 and had received combination pills for regularization of periods for three cycles, a year back. Her periods prior to this episode were regular lasting for 6–7 days but were not heavy. There was no history of easy bruisability in either her or her family. There was no history suggestive of pelvic inflammatory disease in the form of fever, lower abdominal pain or excessive vaginal discharge. Patient gave no history of

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sexual contact. On examination there was gross pallor, but no petechial/purpura spots, hirsutism, obesity, thyromegaly or lymphadenopathy. Systemic examination was normal. Initial laboratory parameters evaluated at admission included a complete blood count, urine pregnancy test, thyroid profile and blood sugar. These were normal apart from severe normocytic–normochromic anemia. Ultrasound showed a normal size uterus with endometrial thickness of 4 mm and a clot in the cervix. The ovaries were not cystic or enlarged. Progesterones (norethisterone 10 mg qid) along with natural conjugated estrogens 2.5 mg qid were commenced orally. Additional test were done the following day as she continued to bleed and these included a coagulation profile, peripheral blood smear, Factor VIII study, hepatic and renal function tests. These too were normal. Following a hematological consultation, she was started on supportive management, with parental anti-fibrinolytic agents (ethamsylate 250 mg qid im) and two units of fresh whole blood, two units of fresh frozen plasma and two plasma platelet fraction. On the third day, the patient showed features of shock with restlessness, tachycardia (PR 110/min), and hypotension (BP 80/40 mmHg), developed drug induced emesis and started passing large clots per vaginum. Repeat ultrasound again showed a doubtful blood clot in the cervix with no other abnormalities. She was taken up for a pelvic examination under anesthesia and found to have a cervical polyp $2.5 \times 2.5 \times 1.5$ cm size, with a broad base, distending the external os but not protruding into the vagina. This was electro-cauterized at its base and sent for histopathology. A gentle curettage was done which gave scanty endometrium. The patient showed a dramatic improvement following surgery and was discharged following 24 h observation. Histopathology revealed it to be a myomatous polyp was secretory

endometrium. She has been having regular cycles since surgery and is on no medication at present.

Discussion and Conclusion

Local causes of adolescent menorrhagia are uncommon, accounting for 5–7 % of all cases of severe menorrhagia in various studies. Consequently in a pubescent or adolescent girl with no prior history of sexual contact, cultural sensitivity often is a factor for not conducting a pelvic examination. However a local cause must be excluded if there is no response to expectant treatment for anovulatory bleeding, pregnancy being excluded and the coagulation profile being normal. This is a rare presentation of adolescent menorrhagia and we have found no similar case reported in the literature reviewed so far.

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