

Congenital Uterine Cervical Stenosis Causing Primary Amenorrhea. A Case Report

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Abstract

Congenital genital tract obstruction such as uterine cervical stenosis is a rare cause of primary amenorrhea. It poses management challenges as advanced radiological evaluation for proper definition of genital tract anatomical borders and required surgical care by experienced gynaecologist are key for a successful outcome.

Keywords: Primary amenorrhea; Genital tract; Obstruction; Congenital cervical stenosis; Cryptomenorrhea

1. Introduction

Primary amenorrhea resulting from outflow tract obstruction of genital tract may be due to various causes. Though the most common reason is an imperforate hymen followed by transverse vaginal septum and rarely cervical agenesis [1], cervical stenosis which could be congenital or acquired can also occur. The stenosis can be complete or partial obliteration of the cervical canal which can occur at the level of the internal, external cervical os or the entire length of the cervical canal. In the case of congenital cervical stenosis, at menarche, the menstrum continues to be held back in the endometrial cavity, forming haematometria and as well, resulting in primary amenorrhea. On the other hand, it will impair the access to the endometrial cavity, thus leading to failure to perform the intended hysteroscopic procedure or complications such as uterine perforation, cervical laceration, or the creation of a false passage [2,3]. Although there is no consensus on the definition of cervical stenosis, it could be defined as a cervix with an obliterated cervical ostium and/or cervical canal that requires particular maneuvers for the introduction of the hysteroscope in order to access the uterine cavity [4]. According to Baldauf's definition, cervical canal stenosis occurs when the cervical canal does not allow the passage of a 2.5 mm Hegar cervical dilator[5], while external cervical os (ECO) stenosis has been defined as when the diameter of the ECO is less than 4.5 mm [6]. The patient whose case is reported had internal cervical os (ICO) stenosis with complete obliteration of the os preventing outflow of monthly bleed. The true incidence of cervical stenosis cannot

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be estimated because most patients are asymptomatic and this condition is only diagnosed in those with primary amenorrhea, scanty menstruation with haematometria due to partial stenosis or in those with indication of evaluation of the uterine cavity. The stenosis can present challenges in achieving successful intrauterine procedures.

Operative hysteroscopy has been shown to have the highest success rate, particularly in cases of severe cervical stenosis, and is currently considered the gold standard for managing this condition. Despite the availability of miniaturized instruments that have made the management of cervical stenosis more feasible, it remains a complex task, even for experienced hysteroscopists [4]. In low and middle income countries where there is limited availability of both endoscopic facility and skill, the challenges in achieving successful intrauterine manipulation is even more tasking as the surgical procedure is still via a combination of laparotomy and blind cervical dilation using hegar's dilator/uterine sound through the vaginal route. The surgical procedure used for our patient was laparotomy/hysterotomy in combination with cervical dilation using hegar's dilator/uterine sound through the vaginal access due to limited endoscopic facility and the skill. Some cases of primary amenorrhea or scanty menstrual flow with haematometria from complete or partial cervical stenosis have been reported [7-10].

In this case report, we describe a 22 year old single nullipara who presented with primary amenorrhea due to complete congenital internal cervical os stenosis and had surgical canalization with good outcome.

2. Case report

Miss NA was a 22 year old single nullipara who presented to the gynaecology clinic of Enugu State University Teaching Hospital, Parklane, Enugu, with complaints of not having her first ever menstruation. This has been a major cause of concern in the entire family as her younger female siblings have all started menstruating including her 14 year old younger sister. There was no similar history of delayed menstruation in the family. She had normal growth pattern including normal breast development like her other siblings and there was no history suggestive of any chronic childhood illness or any condition requiring use of ironizing radiation or cytotoxic drugs. There was no history of weight loss or gain, abnormal hair pattern or acne and there was no expressible galactorrhea. She had no prior history of pelvic surgery. She was sexually active but several pregnancy tests showed negative results. However, about 8 years prior to her presentation, she noticed lower abdominal pain which has been coming on monthly basis. It was like cramps and spans for a period of 4 days and waned. There was initially no associated gastrointestinal or lower urinary tract symptom. About 8 months prior to her presentation, she noticed a swelling on the lower abdomen centrally, this has increased in size gradually with associated lower abdominal pains on urination as well as pains on defecation but there was no other gastrointestinal or lower urinary tract symptoms. For these complaints she presented to the general outpatient clinic of the hospital where abdominopelvic ultrasound reported bulky uterus with endometrial fluid collections about 27 ml and a left adnexal mass (figure 1) and she was then referred to the gynaecology clinic for expert evaluation. At presentation, she was in no obvious painful distress. She was afebrile with a temperature of 36.6°C. She was anicteric and not pale. Her weight was 45kg and height was 1.62m and body mass index of 17.14kg/m². Her blood pressure was 120/70mmHg and her pulse rate was 70 beats per minute. Her breast examination showed tanner stage 5. Her chest was clinically clear. The abdomen was full with a suprapubic bulge about 14 weeks size and not tender. Vagina examination showed normal external genital development. Cervix was grossly healthy looking with normal appearing external cervical os. Uterus was bulky about 14 week size. There was a left adnexal mass. There was no cervical motion tenderness. Assessment was primary amenorrhea possibly due to uterine cervical stenosis. She was counseled on the findings and the required surgical procedure. The abdominopelvic ultrasound showed enlarged uterus measuring 14.5 x 5.6 x 5.5cm with echo-rich collection of 27ml within the endometrial cavity and right adnexal complex mass with conclusion of haematometria secondary to obstruction at the level of internal cervical os (Figure 1). Magnetic resonance imaging made impression of cervical stenosis at the level of internal cervical os with haematometria with bilateral haematosalpinges more on the left fallopian tube (Figure 2). Her haematological reports were normal (figure 3). Her kidney function and Liver function tests were normal (figure 4). Diagnosis of cervical stenosis was then made. She was counseled on her condition and the proposed laparotomy and possible surgical canalization of the cervix. Informed written consent was obtained for the surgery. She had anaesthetic review and was certified fit for surgery. She subsequently had exploratory laparotomy and hysterotomy with evacuation of 30 ml of menstium from the endometrial cavity while graded Hegar cervical canalization was done with visualization of the hegar in the endometrial cavity (figures 5 to 8). Size number 10 silicone and Foley catheter were passed through the opened internal cervical os and left in situ till her first ever menstruation commenced on the 28th September, 2025.

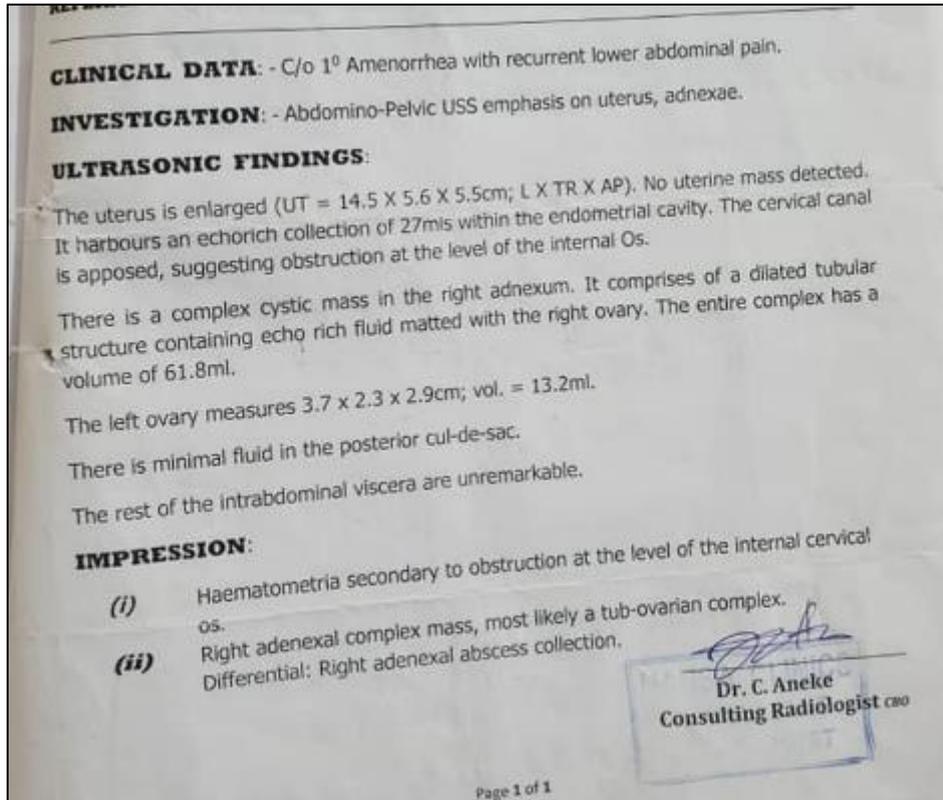


Figure 1 Abdominopelvic ultrasound scan

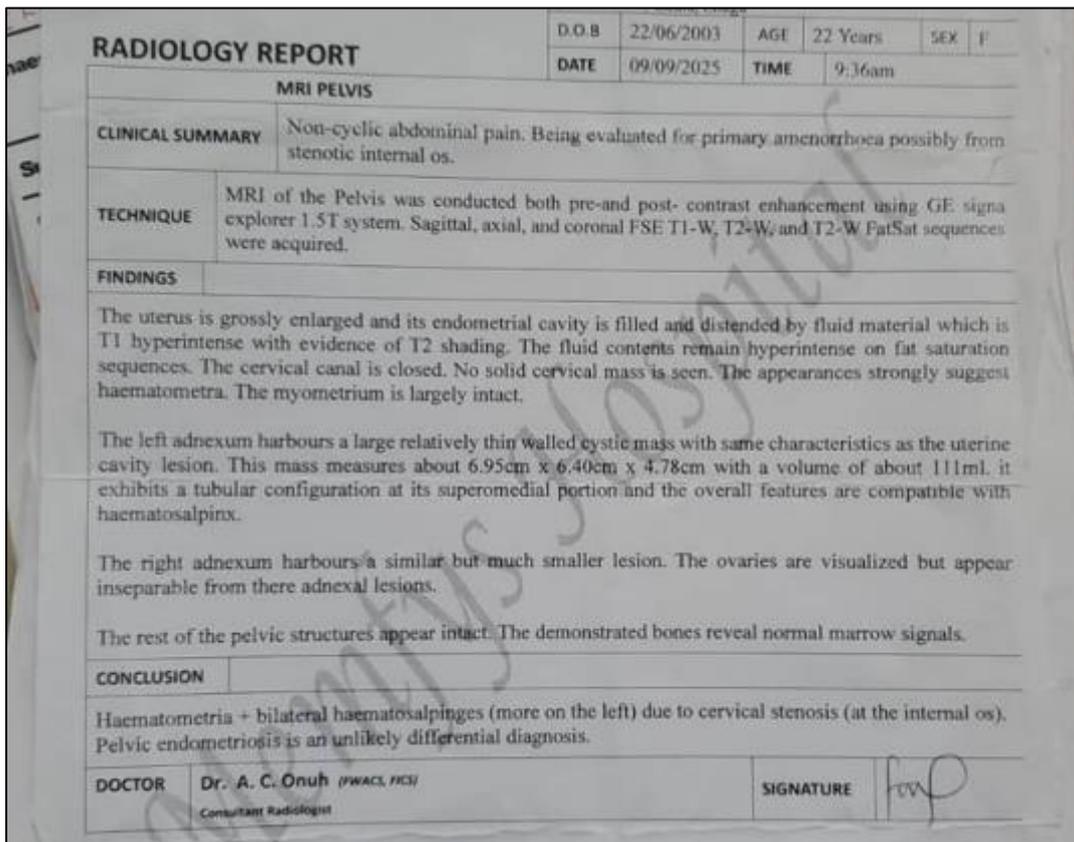


Figure 2 Abdominopelvic Computed Tomography scan

Patient ID: DG-06896-25 Sex: Female Age: 22
 INVESTIGATION(S): FBC

Laboratory Report

PARAMETER	RESULT	UNIT	REF. RANGE
FULL BLOOD COUNT			
Hb	14.5	g/dl	F: 12.0-16.0 M: 13.0-16.0
Hct	44	%	F: 35.0-48.3 M: 39.0-54.3 C: 25.2-45.1 (%)
WBC TOTAL	7700	/mm ³	AC: 4300-10000 C: 4000-10000
NEUTROPHIL	68	%	F: 31.0-62.0 M: 24.0-64.0 C: 30.0-60.0 (%)
LYMPHOCYTE	26	%	F: 36.0-50.0 M: 17.0-40.0 C: 15.0-35.0 (%)
EOSINOPHIL	5	%	0.0-6.0
MONOCYTE	1	%	0.0-10.0 (%)
BASOPHIL	0	%	0.0-1.0
PLATELET	230	/mm ³	150-400
MCV	92	fL	80.0-101.0 (f)
MCH	26	pg	27.0-34.0
MCHC	31.3	g/dl	27.0-36.0 (g/dl)
RDW CV	13.9	%	~
RDW SD	41.5	fL	~

FBC: Samples from this lab are for transfusion use that is suitable for use as a starting transfusion after an external physical exam is performed and suitable for transfusion and distribution of the results.

Figure 3 Full blood count

Date Printed: 9/22/2025

PARAMETER	RESULT	UNIT	REF. RANGE
KIDNEY FUNCTION TEST			
CREATININE	95	mmol/L	Adult: 61 - 136 Adolescents: 20 - 100 Infant: ~100
UREA	3.2	mmol/L	< 6.0
SODIUM	140	mmol/L	133 - 145
POTASSIUM	3.8	mmol/L	3.5 - 5.0
CHLORIDE	101	mmol/L	95 - 110
BICARBONATE	24	mmol/L	18 - 30

NOTES:
 (1) Patient disease processes values may be abnormal.
 + = Abnormal finding
 = = Abnormal range
 - = Abnormal result
 (2) Patients are advised to take results with clinical values to the physician for further investigation and follow up.

PARAMETER	RESULT	UNIT	REF. RANGE
LIVER FUNCTION TEST			
BILIRUBIN (TOTAL)	0.3	mg/dl	< 1.1
ALBUMIN (G%)	0.1	mg/dl	>= 0.0
ALANINE TRANSFERASE	29	U/L	< 60
ASPARTATE TRANSFERASE	46	U/L	< 45
ALUMINE PHOSPHATE	198	U/L	< 270

NOTES:
 (1) Abnormal findings may be:
 + = Abnormal finding
 = = Abnormal range
 - = Abnormal result
 (2) Patients are advised to take results with clinical values to the physician for further investigation and follow up.

Med Lab Scientist **VALIDATED BY DR. C. N. OMEJE (DCMLS)** Quality Control Sc.

Figure 4 Serum Electrolyte urea and creatinine and Liver Function Tests

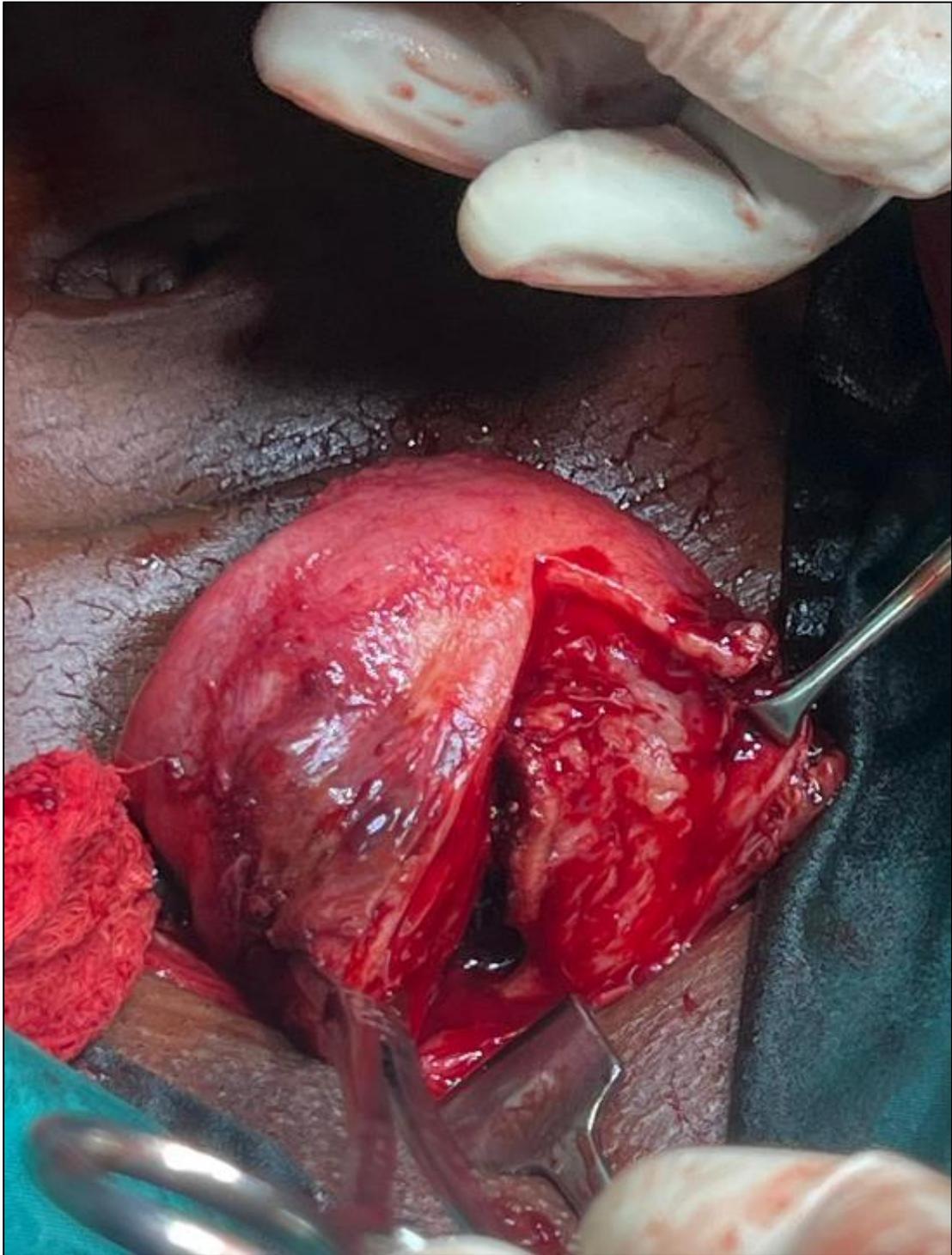


Figure 5 Intra-operative findings

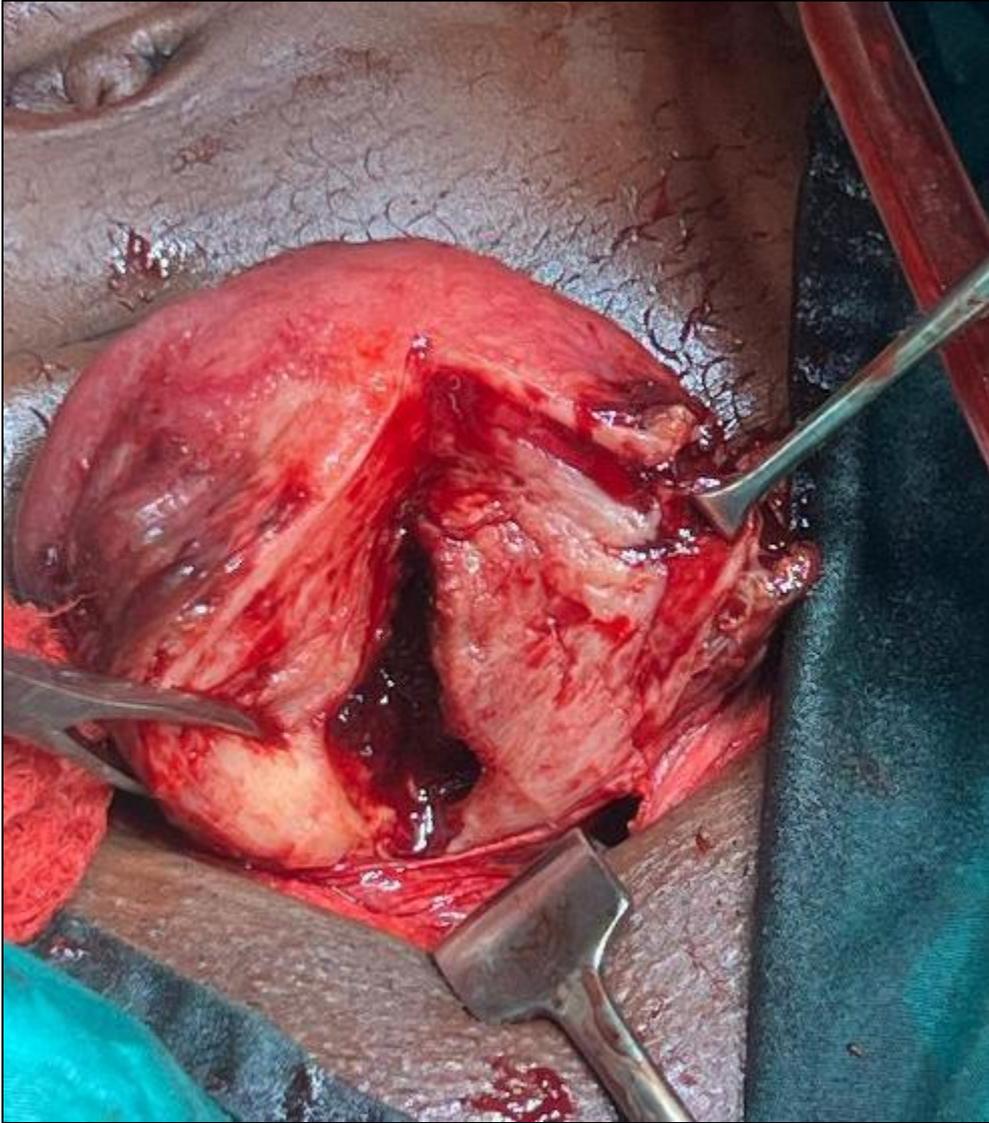


Figure 6 Intraoperative findings

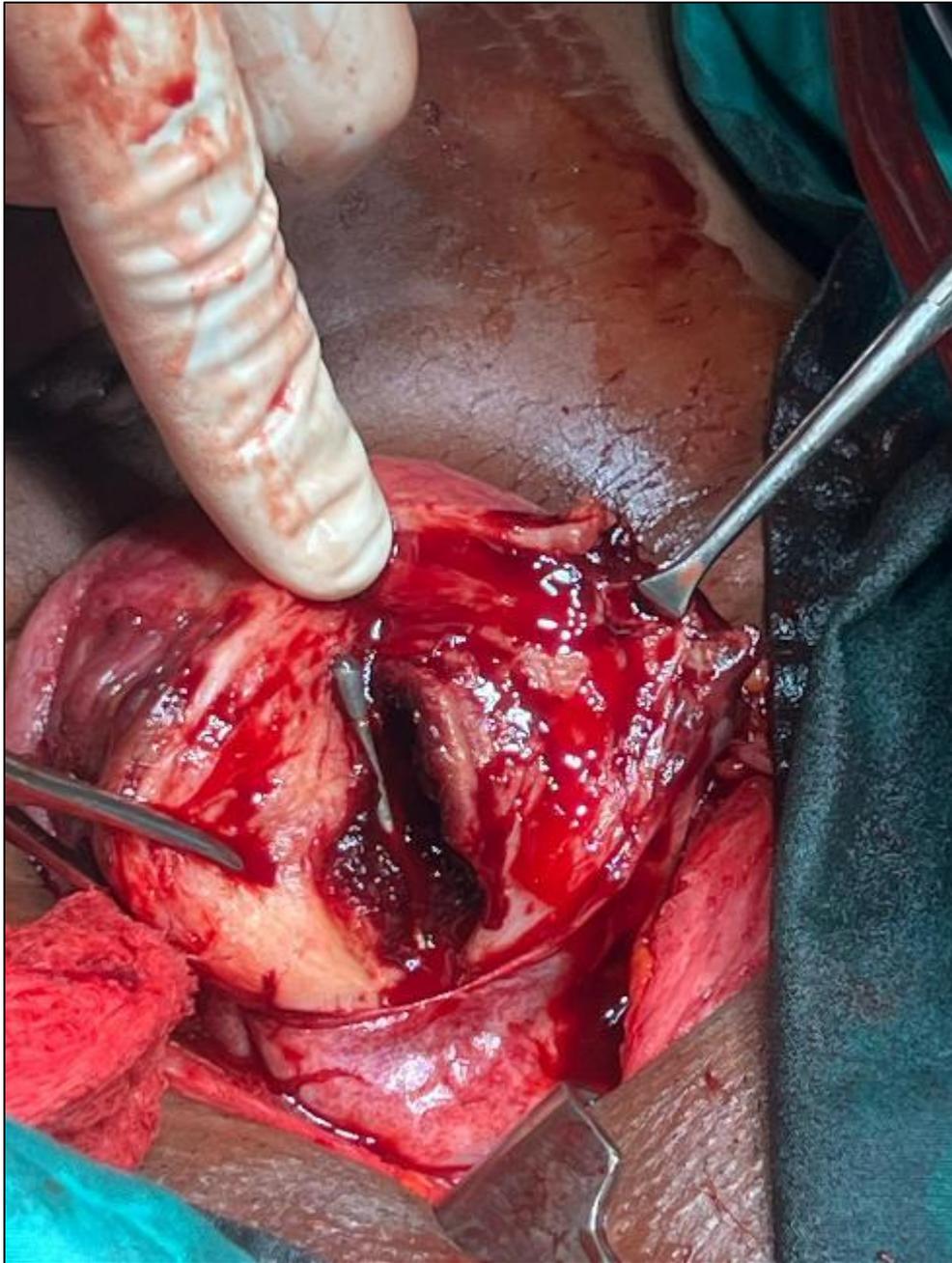


Figure 7 Intraoperative findings



Figure 8 Intraoperative findings

3. Discussion

Primary amenorrhea with haematometra is a common finding in the cases of either congenital or acquired genital tract obstruction. Primary amenorrhea due to imperforate hymen and transverse vaginal septum are well known but cervical stenosis though mentioned in literature is a rarity. Both congenital and acquired cervical stenosis have been reported. While congenital type is more rare [8,10], and occasionally co-existing with congenital uterine anomaly [9], acquired cervical stenosis seems to be more common, occurring occasionally as a complication of cervical conization [5], Manchester repair, caesarean section [11], vesicovaginal fistula repair [13,14]. Primary amenorrhea is commonly seen in congenital cervical stenosis that involved the complete obliteration of the cervical canal while secondary amenorrhea is seen commonly with the acquired type. Our patient had complete obliteration of internal cervical os with primary amenorrhea. According to the classification proposed by Bettocchi in 2016 that recognizes four types of cervical stenosis depending on the structure or structures affected (ECO, cervical canal, ICO) [12]. Our patient had type III cervical stenosis as the level of obstruction was at the level of internal cervical os. This was a complete obliteration of the internal cervical os leading to accumulation of menstrual blood in the endometrial cavity called haematometria. In some occasions, complete or a partial cervical stenosis, the endometrial collections can track down the fallopian tubes leading to haematosalpinges [7]. This was seen in our patient. In Indonesia, Sari and his study group reported cervical stenosis in a 16 year old adolescent [10]. Though there was haematometra as seen in our patient, there was no primary amenorrhea as stenosis was partial. Hence, there was monthly scanty menstrual flow and the stenosis located at the level of the external cervical os. These were different from the findings in our patient where the stenosis was at the level of the internal cervical os and complete, leading to complete absence of menstruation. However, both cases were congenital type of cervical stenosis. In Hunan Province, China [9], 2 cases of congenital cervical stenosis were reported. Although the age of the adolescents reported were far younger, 12 and 14 years compared to our patient that was 22 years, the pattern of abdominal pains at presentations were essentially the same. While they had partial stenosis and at the external cervical os with light menstruation and haematometria, our patient had complete obliteration of the internal cervical os with primary amenorrhea. The partial external cervical os stenosis could be due to the associated presence of uterine anomaly, complete uterine septum. Such uterine anomaly was not seen in our patient. Similar

primary amenorrhea, haematometria and haematosalpinges from a genital tract obstruction was reported in India by Bal and the colleagues [7]. While the obstruction was due to cervical agenesis, our patient had stenosis of the cervix. Although similar findings lower abdominal pains, amenorrhea, hypomenorrhea and haematometria due to cervical stenosis were reported by Santosh et al in India [13], Lilungulu et al in Tanzania [14] and Chrysostomou et al in South Africa [11], the cervical stenosis were all acquired and occurred due to complications of caesarean sections and vesicovaginal fistula repair. The amenorrhea was secondary type. This was dissimilar to congenital cervical stenosis with primary amenorrhea seen in our patient.

4. Conclusion

Congenital uterine cervical stenosis is a rare form of genital tract obstruction and poses management challenges. The gold standard treatment is hysteroscopy. However, in low resource setting, where endoscopic skills and facilities are limited, a combination of laparotomy and vaginal route in canalizing the cervical stenosis could suffice and this approach was used for our patient with good outcome.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The authors declare that they have no conflicts of interest.

Statement of ethical approval

Ethical approval was obtained according to institutional protocols. Approval number: ESUCOM/FBMS/ETR/2026/ 010. Privacy and confidentiality of the patient was fully observed.

Statement of informed consent

A written informed consent was obtained from the patient for publication of this case report.

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