Chronic Pelvic Pain Secondary to Adenomyosis in Mayer– Rokitansky–Küster–Hauser Syndrome

Philip Judson M. de la Vega, MD and Madonna Victoria S. Calderon-Domingo, MD, MBA, FPOGS, FPSRM, FPSGE
Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology,
Philippine General Hospital, University of the Philippines Manila

Abstract

In Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome, the development of the uterus and some parts of the vagina is either completely absent or reduced. It is a rare congenital anomaly, and affects one in 4,000–5,000 female births and commonly presents as primary amenorrhea. Approximately 6% - 10% of these patients with MRKH syndrome report persistent pelvic pain, which may be attributed to the presence of myomas, endometriosis, adenomyosis or hematometra caused by a functioning endometrial tissue in a uterine remnant. This paper presents the case of a 37 year old nulligravid who experienced severe cyclic hypogastric pain, and was subsequently diagnosed with MRKH syndrome with adenomyosis. Clinical evaluation and definitive management of the index case are discussed.

Key words: Mullerian duct failure, adenomyosis, pelvic pain

Introduction

Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome is a congenital anomaly caused by the aberrant development of the Müllerian ducts.[1] The Müllerian ducts normally differentiate into the uterus and the upper one-third of the vagina, during early fetal development. Thus, in MRKH syndrome, the development of a well-formed uterus and upper part of the vagina is either completely absent or reduced. The MRKH syndrome affects one in 4,000–5,000 female births and commonly presents as primary amenorrhea.[2,3] In about 10% of these cases, functional endometrial islands may be present, which cause cyclic pain symptoms.[4]

On the other hand, uterine adenomyosis is a benign condition characterized by the presence of ectopic endometrial glands and stroma, two high-power fields below the basement membrane of the endometrium.[2,5] Diffuse uterine enlargement is its defining feature, while some people may also have localized nodular lesions. Menorrhagia, dysmenorrhea, and cyclic pelvic discomfort are the most common clinical manifestations.

There are very few published reports of adenomyosis occurring in the uterine buds of patients with MRKH syndrome.[2,3] This paper presents the case of a 37 year old nulligravid who experienced severe cyclic hypogastric pain and subsequently diagnosed with MRKH syndrome with adenomyosis. Evaluation and management of the patient were likewise discussed.

The Case

This is a case of a 37-year-old nulligravid, middle-aged Filipino woman who consulted with a chief complaint of severe hypogastric pain. She has been married for 16 years, unable to conceive, and has an adopted son. The patient was a known hypertensive since 2021 and underwent cholecystectomy for calculous cholecystitis in November 2023. She has no familial history of any malignancy or kidney disease. She never experienced menarche, and her

*For correspondence: philipjm.dlv@gmail.com

first coitus was at 16 years old. She has a non-promiscuous sexual partner, with sexual activity occurring at most twice a year, with reports of deep dyspareunia. She denies use of any contraceptive, nor history of sexually transmitted infections.

She noted development of breast buds and pubic hair at 11 years old, but did not experience menarche. The patient claimed she was not educated about menstruation and was told by friends that her menstruation might have been just delayed. At 14 years old, she started to experience infrequent crampy pelvic pain lasting for 2-3 days, about 2-3x a year, with an intensity of 5/10, which spontaneously resolved without any medications.

Eight years prior to consultation, she visited a gynecologist to seek help regarding her recurrent pelvic pain. Transvaginal ultrasound showed an infantile uterus with indistinct endometrium, myoma uteri (right lateral subserous measuring 2.7 cm x 2.4 cm), and normal ovaries. Upper abdomen ultrasound showed cholesterolosis, polyp and encrusted stone. The patient was reassured, and no further plans or workups were done.

Six years prior to consultation, the patient experienced monthly, severe, crampy epigastric pain and bloating, which became generalized and finally localized to the right lower quadrant. The pain radiated to the thighs and lasted for four to 7 days. A repeat transvaginal ultrasound revealed similar findings. About a month prior to consultation, the patient visited the emergency room for severe abdominal pain and was subsequently referred to an OB-GYN for evaluation of amenorrhea and myoma. Upon consultation with the OB-GYN the following month, she underwent several tests, including a repeat transvaginal ultrasound and hormone assays. She was later referred to hepatobiliary surgery for a cholecystectomy and to a Reproductive Endocrinology and Infertility (REI) clinic for further operative planning.

On physical examination, patient was 142 cms tall, and weighed 65 kg (BMI 32.23 kg/m^2). Her vital signs were within normal limits. Tanner staging was 5 for both breast and pubic hair, compatible for her age. There were no breast masses and the rest of the systemic findings were normal. Speculum examination revealed pink and smooth vaginal walls, with no cervix visualized. On internal examination, she had a smooth, shortened (approximately 5 cms) nulliparous vagina that ends in a blind pouch, no palpable cervix, corpus was globular and directed more to the right, and no adnexal masses nor tenderness.

Results of imaging studies included the following:

Transvaginal and transabdominal ultrasound (Figures 1 to 4): Consider bilateral rudimentary hemiuteri with upper vaginal agenesis and lower uterocervical dysplasia: right hemiuterus with non-communicating rudimentary horn and adenomyosis with adenomyoma; left hemi-uterus with non-communicating rudiment horn; thin endometrium (right rudimentary horn); normal right ovary; left ovarian cyst, consider physiologic cyst.

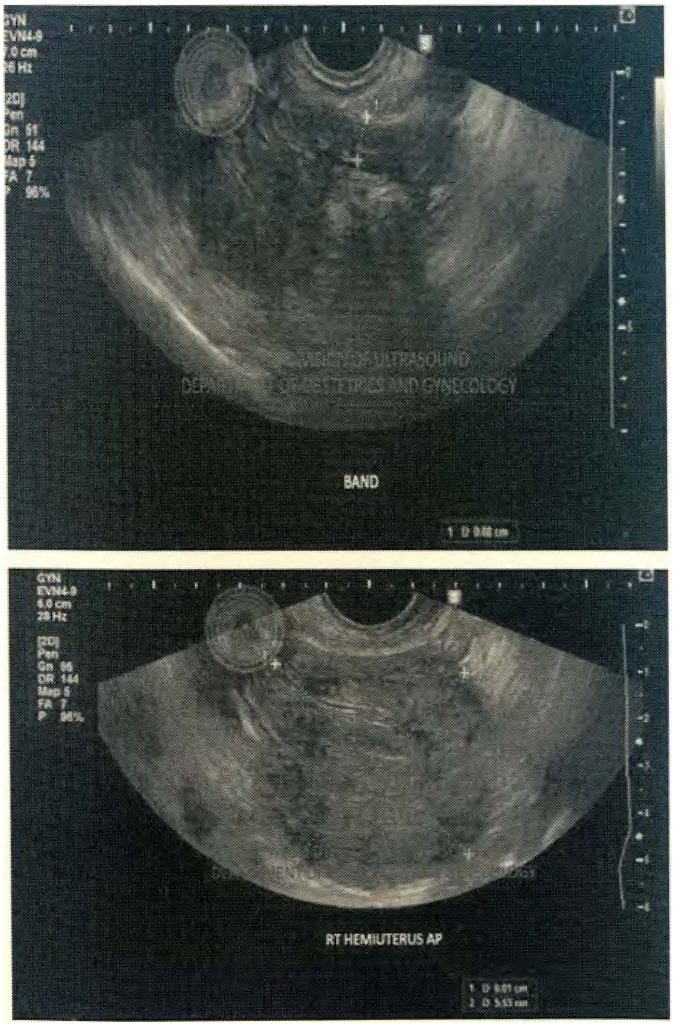
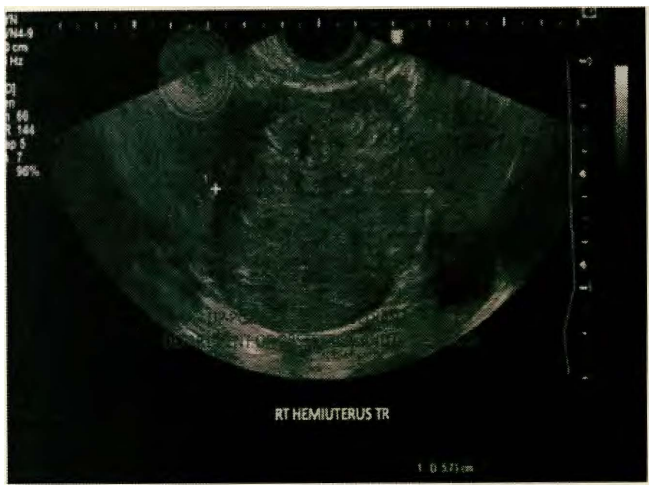


Figure 1. Transvaginal ultrasound: There is no normal cervix visualized. Instead, there is a hypoechoic band with 0.9 cm thick and 3.1 cm long and seems to be connected to a left rudimentary uterus.



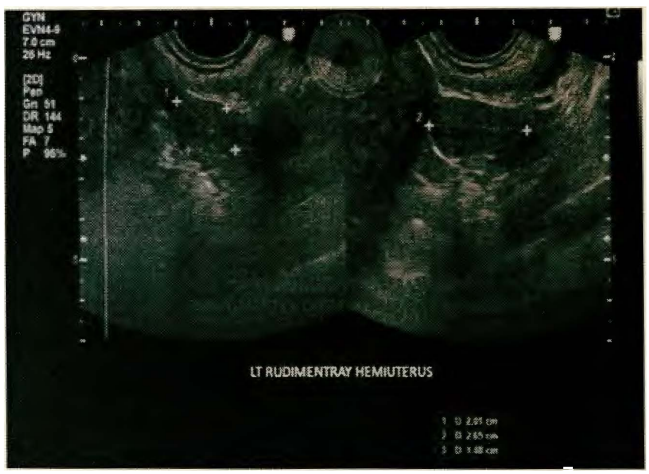


Figure 2. Transvaginal ultrasound: The right hemiuterus is globular with heterogeneous echopattern measuring 6.0cm x 5.7cm x 5.5cm. The myometrium is asymmetrically thickened measuring 5.5 cm anteriorly and 0.8 cm posteriorly with hyperechogenic islands and posterior acoustic shadows. The uterine serosa is smooth and intact. The uterine cavity is poorly defined. The lower third of the endometrium is uniform, hyperechogenic measuring 0.2 cm. The upper endometrium is indistinct.

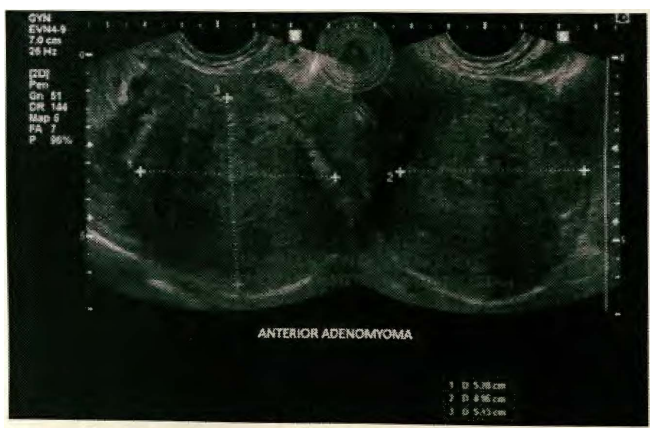
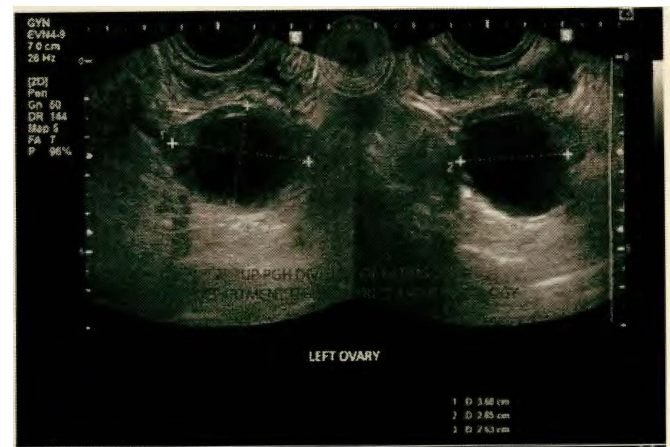


Figure 3. Transvaginal ultrasound: There is an ill-defined heterogeneous myometrial mass measuring 5.3 cm x 5.0 x 5.1 cm at the anterior myometrium.



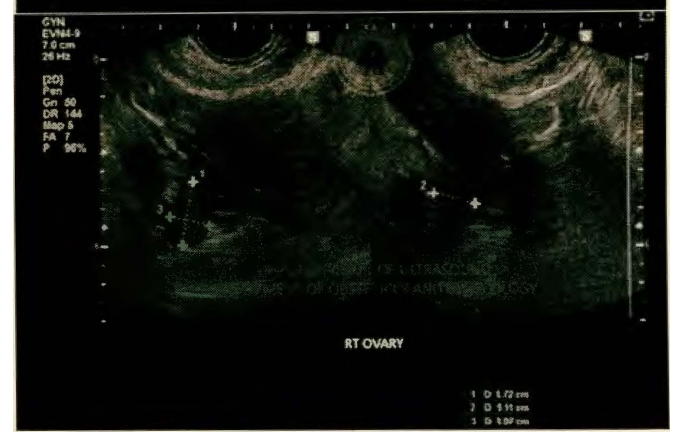


Figure 4. Transvaginal ultrasound: Within the left ovary is a unilocular cyst measuring 3.7cm x 2.9cm x 2.6cm (volume: 14.6cc) with low-level echo fluid within. There are no solid areas or papillary excrescences seen. There is normal ovarian stroma at the periphery. The right ovary is normal and measures 1.7cm x 1.1cm x 1.1cm (volume: 1.1 cc).

Whole abdomen ultrasound: Gallbladder polyp vs encrusted stones; normal ultrasound of the liver, spleen, pancreas, aorta and paraaortic areas, kidneys and urinary bladder.

MRI of the pelvis (Figures 5 to 7): ESHRE/ESGE U6C4V4 classification of female genital tract anomaly; Uterine adenomyosis; Ovarian cysts, left.

The initial diagnosis was Primary amenorrhea secondary to Mullerian anomaly, Upper vagina agenesis with uterocervical dysplasia. Adenomyosis with adenomyoma, Hypertension stage I, controlled, Impaired fasting glucose, Calculous cholecystitis.

The patient eventually underwent open cholecystectomy and intraoperative cholangiography. With persistence of severe pelvic pain despite medical management, she was advised to undergo hysterectomy. Intraoperatively (Figures 8 to 10), the right rudimentary uterus measured 9cm x 8cm

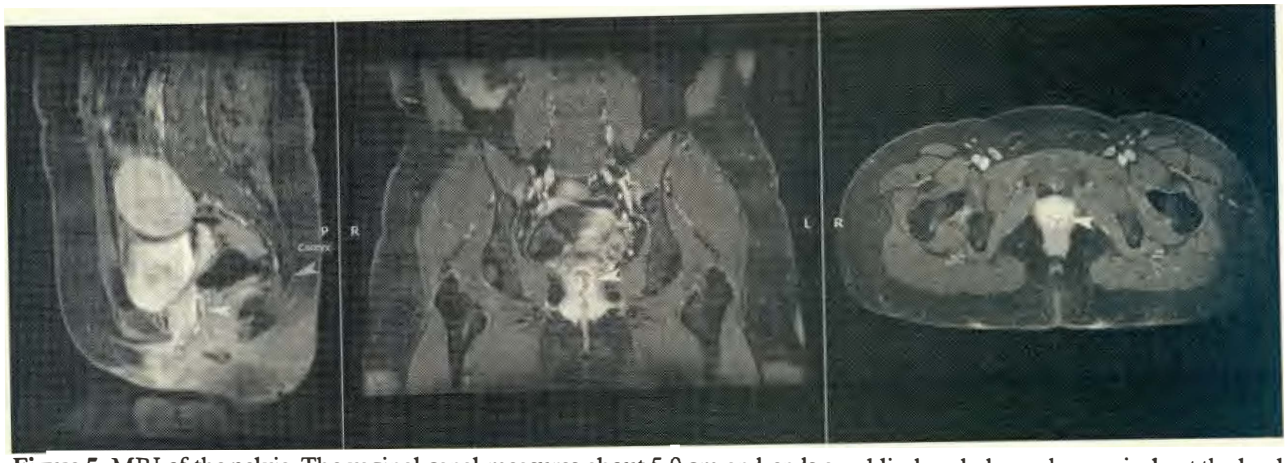


Figure 5. MRI of the pelvis: The vaginal canal measures about 5.0 cm and ends as a blind-ended pouch superiorly at the level of the coccyx. Its patent lower aspect remains distinct from the urethra along their course until the perineum.

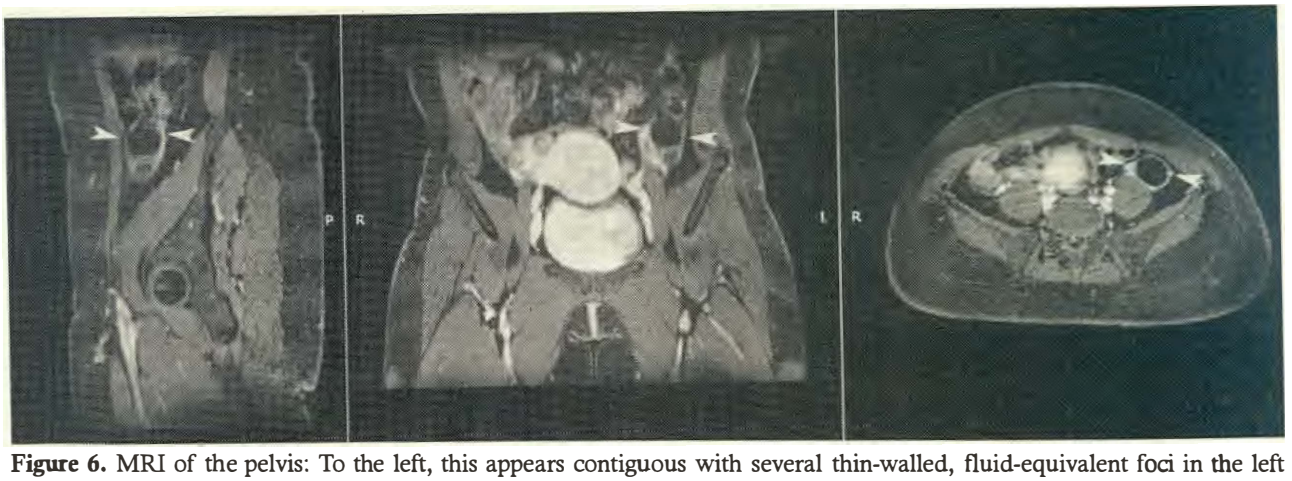


Figure 6. MRI of the pelvis: To the left, this appears contiguous with several thin-walled, fluid-equivalent foci in the left adnexal region. The largest cyst measures about 2.7cm x 2.5cm x 2.9cm (CC x T x AP). No definite canal is detected.

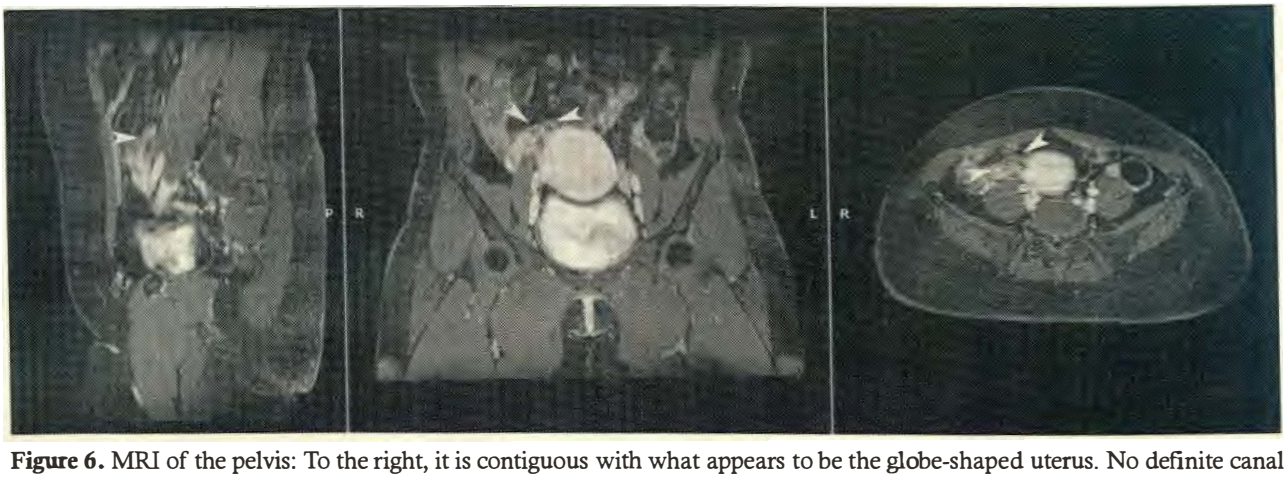


Figure 6. MRI of the pelvis: To the right, it is contiguous with what appears to be the globe-shaped uterus. No definite canal is seen. The endometrial canal is not clearly identified. The uterus itself measures 8.4cm x 7.1cm x 6.5cm (CC x W x AP) and exhibits heterogeneous, predominantly T1W-/T2W-isointense (to muscle) signals with restricted diffusion. Several small foci of T1W-/T2W-hyperintensity are seen within the uterus. No magnetic susceptibility artifact identified.

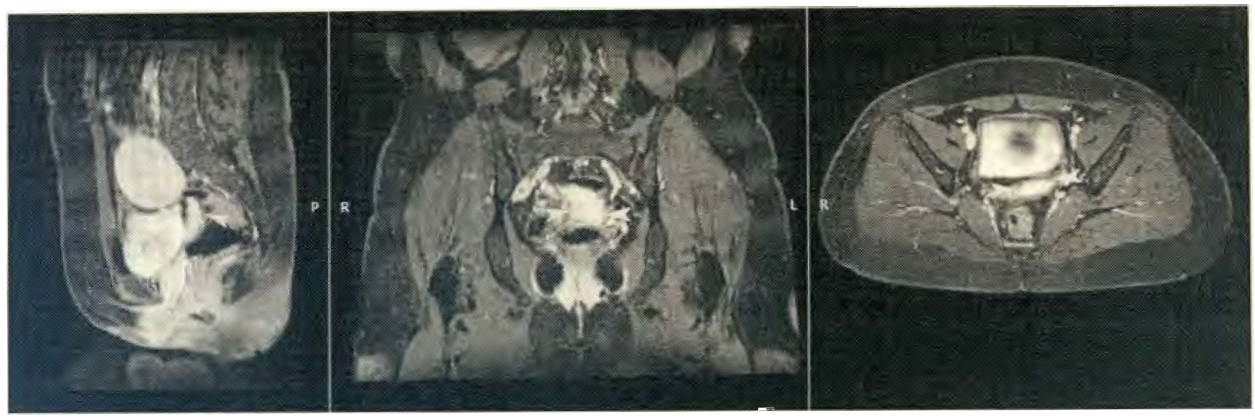


Figure 7. MRI of the pelvis: A nodular, T1W-/T2WI-isointense focus, with no restricted water diffusion and magnetic susceptibility artifact, is seen intimately related to the left posterolateral aspect of the uterus, measuring approximately 1.3cm x 1.6cm (W x AP). This may represent the aplastic cervix.

x 6 cm, while the left rudimentary uterus measured 2.5cm x 2cm x 2cm. Both ovaries and fallopian tubes were grossly normal. There were no powder-burn lesions or adhesions suggestive of endometriosis. Resection of bilateral rudimentary uteri with bilateral salpingectomy was done. The estimated blood loss was 250 ml.

On cut section (Figures 11 - 12), the right rudimentary uterus showed a tan smooth surface with trabeculations and ill-defined hypertrophic swirls of smooth muscle. There was a cavity, but

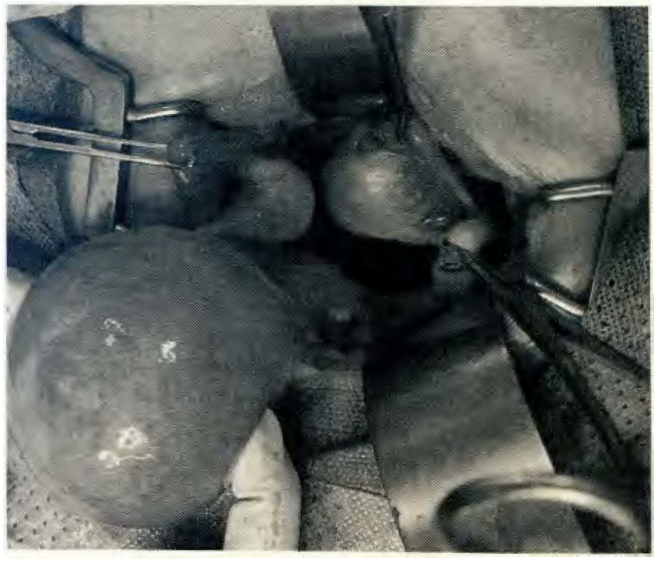


Figure 8. Intraoperative findings: From patient's right to left: right fallopian tube clamped by allis clamp; adjacent to it is the right ovary. Attached to the right ovary is the right uterine remnant (pointed by the gloved index finger); left fallopian tube grasped by babcock clamp; adjacent to it is the left ovary with cystic follicle attached to the left uterine remnant (grasped by babcock clamp).

Figure 9. Intraoperative findings: Adenomyotic right uterine remnant.

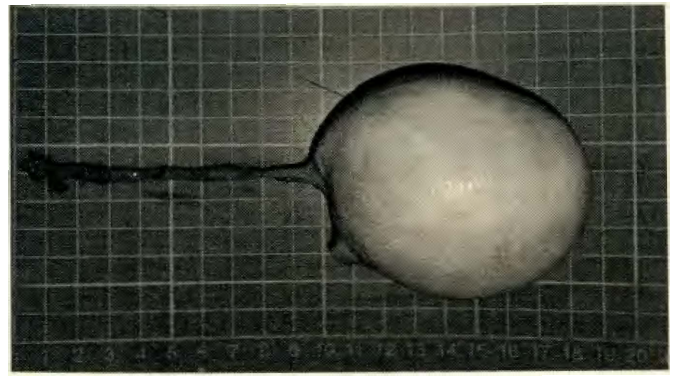


Figure 10. Right rudimentary uterus measured 9cm x 8cm x 6 cm.

no endometrium was identified; the anterior surface measured 6 cm, and the posterior surface measured 1 cm. No areas of hemorrhage nor necrosis were noted. The right fallopian tube measured 8cm x 1cmx 1cm and was grossly normal. The cut section of the left rudimentary uterus showed a tan smooth surface. The left fallopian tube measured 8cm x 1cm x 0.8cm and was grossly normal.

The patient's postoperative course was unremarkable. Histopathology report (Figures 13 - 14) confirmed presence of adenomyosis on the rudimentary uterus.

On follow up at the outpatient clinic, the patient remained stable, with no recurrence of hypogastric pain. The patient and her husband were counseled regarding the use of vaginal dilator therapy for the shortened vagina, to address the dyspareunia.

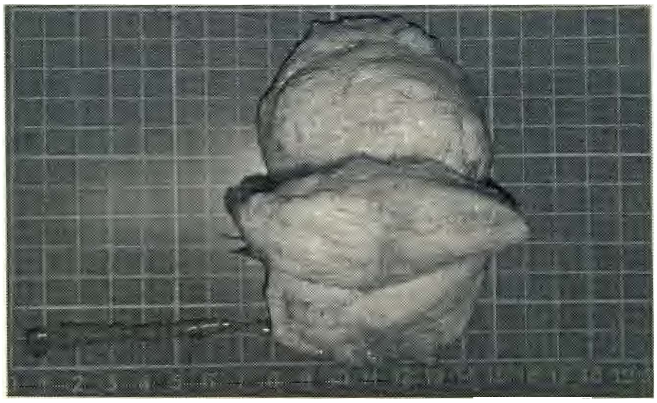


Figure 10. Right rudimentary uterus measured 9cm x 8cm x 6 cm.

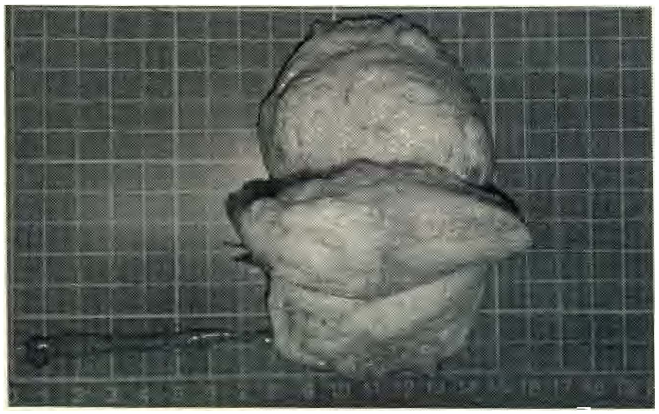


Figure 11. Cut section of the right rudimentary uterus showed tan smooth surfaceswith trabeculations and ill-defined hypertrophic swirls of smooth muscle. There was cavity but no endometrium was seen; anterior surface measured 6 cms, posterior surface measured 1 cm. There were no areas of hemorrhage nor areas of necrosis noted. The right fallopian tube measured 8cm x 1cm and was grossly normal.

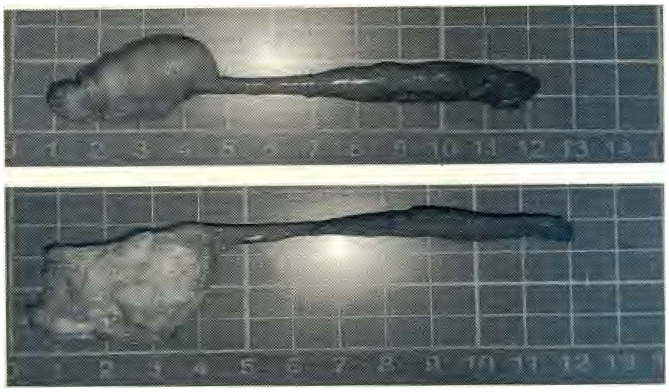


Figure 12. The left rudimentary uterus measured 2.5cm x 2cm x 2cm. Cut section of the left rudimentary uterus showed tan smooth surface. The left fallopian tube measured 8cm x 0.8cm, and was grossly normal.

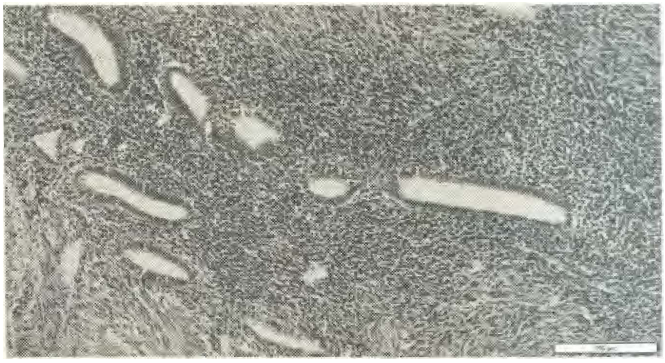


Figure 13. Low power view of the right rudimentary uterus showed presence of endometrial glands and stroma within the myometrium.

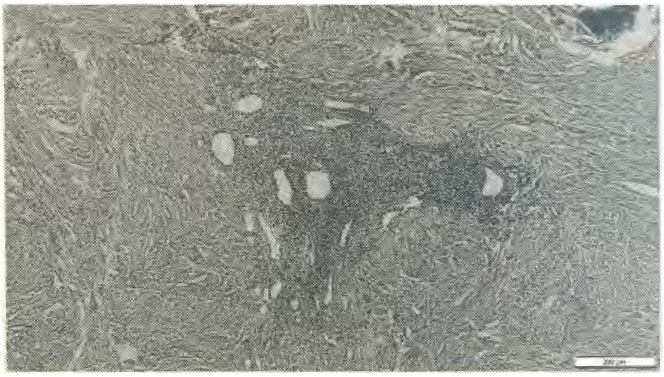


Figure 14. Scanning view of the right rudimentary uterus showed presence of endometrial glands and stroma within the myometrium.

Discussion

The exact cause of Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome remains unknown, although a developmental field malfunction may

be the most plausible explanation. An arrest in Müllerian duct development during the seventh week of embryologic development may be the cause of this condition.[7,8] Although familial cases have been reported, sporadic cases make up the majority of reported cases.[9] The mode of inheritance appears to be autosomal dominant, and some of the genes identified are TBX6, WNT4, HOXA and HNF1B gene.[10]

Vaginal aplasia and normal external genitalia are the main characteristics of Mayer- Rokitansky-Küster-Hauser (MRKH) syndrome. This is because the origin of the uterus, cervix and upper third of the vagina is the Müllerian duct, while the lower two-thirds of the vagina and vulva originate from the urogenital sinus. Other characteristic findings include an absent or extremely primitive uterus, normally developed fallopian tubes, normal and functional ovaries, normally developed secondary sexual characteristics, and an association with skeletal and urinary system anomalies, middle ear abnormalities, and hearing loss.[8] In the index case, the patient's upper vagina did not develop, and she presented with an undeveloped uterus with indistinct endometrium on the right and absent endometrium on the left and no visualized cervix. Consistent with literature, the patient's external genitalia, fallopian tubes, ovaries, and secondary sexual characteristics were all developed normally.

The absence of menstruation at age 15 with normal growth and sexual features is known as primary amenorrhea.[11] Gonadal failure (Turner syndrome) is the most common cause of primary amenorrhea, while the second most common cause is MRKH syndrome.[11] The index case here exhibited primary amenorrhea, but what prompted repeated gynecological consults was her severe crampy pelvic pain.

Between 6% and 10% of these patients with MRKH syndrome report having persistent pelvic pain, which may be explained by the development of concurrent gynecologic pathologies such as myoma or endometriosis,[4,8,12,13] abnormally located ovaries, or a hematometra which may be caused by a functioning endometrial tissue from a rudimentary uterus.[14,15,16] Other patients may also have endometriotic ovarian cysts.[17,18,19,20] The chronic pelvic pain lasts for more than six months and is often associated with negative behavioral, cognitive, emotional and sexual implications.[21,22,23] In this case, the patient was noted to have adenomyosis on the right rudimentary uterus, which caused her severe pelvic pain.

While ultrasonography may be the preferred initial imaging study when assessing patients who may have MRKH syndrome, MRI would be best considered for this case, especially when assessing the etiology of concurrent pelvic pain.[18,24,25] Laparoscopy is another excellent option, since it can both be diagnostic and therapeutic in patients with MRKH syndrome presenting with a pelvic mass.[26]

A point of interest in this case is the development of adenomyosis on the right rudimentary horn. In theory, uterine adenomyosis usually originates from the endometrium's basal layer, which implies that the uterine mucosa directly invades the uterine musculature to cause adenomyosis.[12] However, there have been reported cases where adenomyosis was not associated with a functional endometrial cavity. Adenomyosis in situ may occur as a result of stromal cell metaplasia influenced by autocrine or paracrine mechanisms, which function like middlemen between genetic, immunologic, and endocrine impacts. Another clinical aspect that supports coelomic metaplasia in this case is the absence of pelvic endometriosis implants or lesions despite intact and patent fallopian tubes. Since there was a functional endometrium with no outflow tract, the menstrual blood was expected to follow the path of least resistance into the fallopian tubes (retrograde menstruation) that can predispose to development of pelvic endometriosis. The absence of such may support the metaplasia theory. Another possibility for the formation of adenomyosis without pelvic endometriosis is that the endometrial tissue was isolated and not contiguous with the tubal ostium. However, this could not be ascertained on histopathology as there was marked inflammation and overgrowth of adenomyotic tissue surrounding the tubal ostia.

The smaller left rudimentary horn was also excised due to the possibility of developing adenomyosis in the rudimentary uterus since it has been postulated that a possible cause of the development of adenomyosis in the right rudimentary uterus is coelomic metaplasia.

Adenomyosis and fibroids hardly ever occur in the rudimentary, nonfunctioning uterus.[13] Enatsu,

et al (2000)[7] described the first case of adenomyosis in a woman with MRKH syndrome. A 52 year old Chinese woman with MRKH syndrome was described by Yan and Mok (2002)[12] as having undergone a hysterectomy for uterine fibroids and adenomyosis. A 42 year old Filipina with MRKH syndrome and adenomyosis in both rudimentary uteri was also described by Ramos, et al in 20196, who underwent an abdominal hysterectomy and bilateral salpingectomy.

A multidisciplinary approach with family counselling is essential in cases of MRKH syndrome. Evaluating associated congenital anomalies is crucial, since up to 53% of patients with Müllerian agenesis may have other congenital malformations, particularly affecting the urinary and skeletal systems.[24] Research has shown that renal anomalies occur in 27–29% of these patients, making ultrasound of the kidneys necessary for all individuals with this condition. Skeletal anomalies—such as scoliosis, vertebral arch defects, and wrist hypoplasia—are found in approximately 8–32% of patients. Thus, spine radiography may detect these issues even in asymptomatic individuals. There is also a modestly increased risk of hearing impairment in patients with Müllerian agenesis. In the case presented, patient was screened for urinary tract anomalies by pelvic MRI and ultrasound, scoliosis was screened by doing chest x-ray. The patient and her husband were not desirous of having a child anymore and no other issues were identified.

Treatment of cases similar to the index patient involves resection of the pathologic uterine remnants.[19] Removal of uterine remnant/s usually poses unique challenges due to the distorted anatomy, possible endometriosis resulting in adhesions, and associated urinary tract abnormalities. Another treatment modality described by Srivastava, et al in 2019[20] is the use of repeated ultrasound-guided superior hypogastric plexus blocks for managing chronic pelvic pain in those with functional endometrium or during ovulation.

Conclusion

Adenomyosis is a rare but possible pathology in patients with MRKH syndrome. Primary amenorrhea, pelvic pain, sexual dysfunction, and infertility are commonly seen in women with MRKH syndrome. To guide proper management, a comprehensive assessment utilizing clinical findings and imaging modalities (MRI and ultrasonography) should be conducted. Excision is indicated when pathologies such as adenomyosis or myoma arise from the Mullerian remnants. A multidisciplinary approach is important to ensure the optimum quality of life for patients with this condition.

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