

Bilateral indirect inguinal herniation of ovaries in a young female with Mayer-Rokitansky-Kuster-Hauser syndrome: An exceptional radiological encounter

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ABSTRACT

We report a rare radiological diagnosis of bilateral indirect inguinal herniation of both ovaries in a 15-year-old girl who presented with primary amenorrhea and normal secondary sexual characteristics. Initial pelvic ultrasonography showed the absence of the uterus in the pelvis and both ovaries within the inguinal canals, each with normal follicular morphology. A preliminary magnetic resonance imaging (MRI) examination performed elsewhere was misinterpreted as aplasia of both the uterus and ovaries. Repeat review at our institution, using dedicated high-resolution pelvic MRI sequences, confirmed Mullerian agenesis consistent with Mayer-Rokitansky-Kuster-Hauser (MRKH) syndrome. Both ovaries were located within bilateral inguinal canals, anterior to the urinary bladder, without torsion or infarction. Endocrine evaluation (follicle-stimulating hormone, luteinizing hormone, thyroid-stimulating hormone, prolactin, estradiol, and androgens) was within age-appropriate reference ranges, supporting preserved ovarian function. The patient and guardians were counseled on the risks of torsion and ischemic insult in ectopic ovaries, fertility implications, and the need for timely surgical reduction and fixation. This case highlights a major diagnostic pitfall in MRKH syndrome: Non-visualization of ovaries in the pelvis does not equate to ovarian agenesis. Targeted sonographic and MRI evaluation of the inguinal region is essential to identify ectopic ovaries and guide appropriate counseling and management.

Key words: Canal of Nuck, Inguinal ovary, Mayer-Rokitansky-Kuster-Hauser syndrome, Magnetic resonance imaging, Mullerian agenesis, Ovarian herniation, Primary amenorrhea

Primary amenorrhea with normal secondary sexual characteristics most commonly results from an outflow tract abnormality or Mullerian duct anomaly. Mayer-Rokitansky-Kuster-Hauser (MRKH) syndrome (Mullerian agenesis) is a congenital disorder characterized by aplasia or severe hypoplasia of the uterus and upper two-thirds of the vagina in individuals with a normal 46,XX karyotype and functioning ovaries. The incidence is approximately 1 in 4500 female births, with most cases being sporadic [1].

Ovarian development is typically preserved in MRKH syndrome, as ovaries derive from the genital ridge rather than the paramesonephric (Mullerian) ducts. Pubertal development is usually normal, including thelarche and pubic/axillary hair, with physiologic gonadotropin and estradiol levels [2,3]. Despite preserved ovarian

endocrine function, patients are unable to carry a pregnancy because of uterine absence; therefore, correct imaging diagnosis is essential for accurate fertility counseling.

Ectopic ovarian location is uncommon but clinically important. Inguinal ovaries occur due to persistence of the processus vaginalis (canal of Nuck) and laxity/elongation of the supporting ligaments, allowing adnexal descent into an inguinal hernia sac. Reproductive organs are reported as contents of the hernia sac in approximately 15–31% of inguinal hernias in girls, but bilateral ovarian herniation is distinctly rare, particularly in association with MRKH syndrome [4,5]. Ectopic ovaries are at increased risk for torsion and vascular compromise, and failure to identify them may lead to incorrect counseling regarding ovarian agenesis or complete infertility.

We present an exceptional radiological encounter of bilateral indirect inguinal herniation of both ovaries in a teenager with MRKH syndrome. This emphasizes

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key ultrasound and magnetic resonance imaging (MRI) findings, diagnostic pitfalls, and management implications.

CASE PRESENTATION

A 15-year-old girl presented to the pediatric outpatient department with primary amenorrhea. She had normal growth and developmental milestones. Puberty began at 12 years with progressive breast development and subsequent pubic and axillary hair growth. There was no history of cyclic abdominal pain, prior pelvic surgery, trauma, chronic illness, or exposure to gonadotoxic medications. Family history was negative for delayed puberty, primary amenorrhea, or congenital anomalies.

On examination, the patient had Tanner stage IV breast development with normal axillary and pubic hair distribution. External genitalia were phenotypically normal. No obvious groin swelling was appreciated on routine inspection; the abdomen was soft and non-tender. A limited perineal examination suggested a short/blind-ending vaginal canal. No skeletal deformities were noted, and there were no urinary symptoms suggestive of renal tract anomalies.

Investigations

The diagnostic evaluation of primary amenorrhea begins with exclusion of pregnancy and systematic assessment of endocrine and anatomic causes. In our patient, a urine beta-human chorionic gonadotropin test was negative. Baseline laboratory tests included serum follicle-stimulating hormone and luteinizing hormone to assess gonadotropin status; estradiol to assess ovarian estrogen production; thyroid-stimulating hormone and prolactin to exclude thyroid dysfunction and hyperprolactinemia; and total testosterone (with clinical correlation) to screen for hyperandrogenic states such as androgen insensitivity or congenital adrenal hyperplasia [6,7]. All measured values were within age-appropriate reference ranges, consistent with preserved ovarian endocrine function and an intact hypothalamic-pituitary-gonadal axis.

Given the suspicion of an anatomic outflow tract abnormality, pelvic ultrasonography was performed using both a curvilinear (pelvic survey) and a high-frequency linear (inguinal) transducer. The uterus was not visualized in the pelvis, and a small blind-ending vaginal remnant was suggested. Both ovaries were absent from the expected adnexal locations. However, bilateral ovoid structures with typical ovarian echotexture and multiple peripheral follicles were identified within the inguinal canals, anterior to the urinary bladder. The right ovary measured approximately $3.1 \times 2.0 \times 2.4$ cm, and the left measured $3.0 \times 2.2 \times 2.3$ cm. Color Doppler demonstrated preserved vascularity in both ovaries. No free fluid, adnexal edema, or whirlpool sign suggested torsion.

Pelvic MRI, performed at an external facility, was initially reported as complete aplasia of both uterus and ovaries. The study was reviewed at our institution and complemented with dedicated pelvic MRI sequences. Multiplanar T1-weighted and T2-weighted images, and short tau inversion recovery images, confirmed uterine aplasia and a small vaginal remnant, consistent with MRKH syndrome. Both ovaries were identified in bilateral inguinal canals (indirect herniation), with preserved stroma and normal follicular distribution without hemorrhage, edema, or restricted diffusion indicating torsion or infarction. The ovaries were located anterolateral to the urinary bladder, with no associated bowel herniation (Figs. 1-3). Screening of the kidneys and urinary tract did not reveal renal agenesis, ectopia, or hydronephrosis, and no obvious spinal anomalies were detected on the available field of view.

Based on clinical presentation, laboratory profile, and imaging findings, a diagnosis of MRKH syndrome with bilateral indirect inguinal herniation of both ovaries was established. The patient and guardians were counseled regarding the diagnosis, the elevated risk of torsion in ectopic ovaries, the need for timely surgical reduction and fixation, and future fertility options, including oocyte retrieval and assisted reproduction with gestational surrogacy.

DISCUSSION

MRKH syndrome is among the leading causes of primary amenorrhea in adolescents with normal secondary sexual characteristics. It arises from Mullerian duct failure, producing congenital absence of the uterus and upper vagina with preserved ovarian structure and function [1,2]. MRKH is broadly categorized into an isolated form (type I) and a form associated with other malformations (type II/MURCS association) involving renal, skeletal, or, less commonly, auditory and cardiac anomalies. MRKH requires MRI for comprehensive characterization of uterovaginal anatomy, identifies functional ovarian tissue, and screens for associated anomalies, thereby informing counseling and management [3].

Inguinal adnexal herniation is uncommon in adolescents but is an important diagnostic consideration when pelvic ovaries are not visualized. A patent processus vaginalis (canal of Nuck) creates a potential pathway for ovarian descent into an indirect inguinal hernia sac. Pediatric series report reproductive organs, most commonly the ovary, as hernia sac contents in approximately 15–31% of girls undergoing hernia repair, with a small but meaningful risk of strangulation or torsion [4,5]. In MRKH syndrome, altered pelvic anatomy and elongated or lax suspensory ligaments may further predispose to ectopic ovarian position. Bilateral ovarian herniation in MRKH is exceptional and has been described only in isolated case reports and small series [8,9].

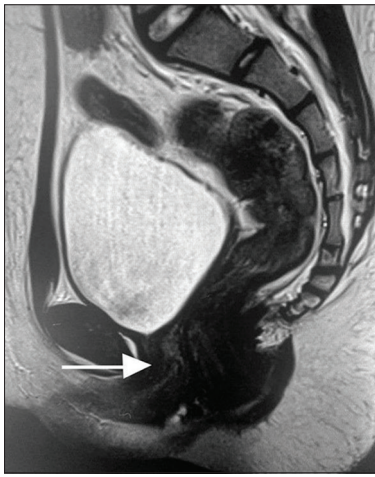


Figure 1: Sagittal T2-weighted imaging showing agenesis of the uterus and upper vagina

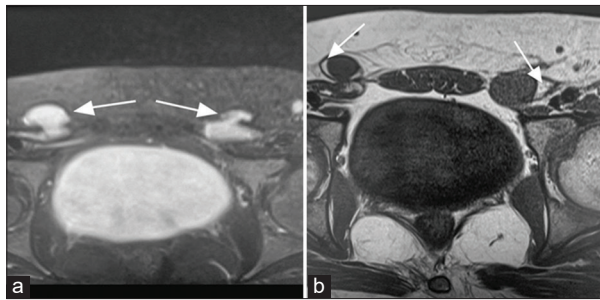


Figure 2: (a) Axial T2-weighted imaging, fat sat, and (b) shows T1-weighted imaging showing bilateral ovaries in the inguinal canal

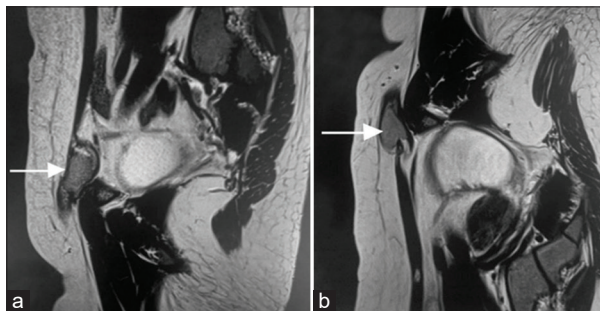


Figure 3: (a and b) sagittal T2-weighted imaging showing the right and left ovaries, respectively, in the inguinal canal

The principal radiologic pitfall highlighted by this case is the erroneous assumption that non-visualization of ovaries in the adnexa implies ovarian agenesis. This occurs with pelvis-limited imaging, omitting the inguinal canals. High-frequency ultrasound is an excellent first-line modality to identify ovarian tissue by its characteristic follicular pattern; color Doppler helps assess viability and may suggest torsion when absent or diminished flow is present. MRI is particularly valuable for problem-solving, providing multiplanar depiction of the inguinal canals, differentiating ovarian tissue from lymph nodes or soft-tissue masses, and identifying complications such as hemorrhagic infarction. In girls with primary amenorrhea, the key clinical-radiologic differential diagnoses include complete androgen insensitivity syndrome (phenotypic female with 46,XY karyotype, absent uterus, and intra-abdominal or

inguinal testes), gonadal dysgenesis (hypergonadotropic hypogonadism with streak gonads), and obstructive outflow disorders (e.g., transverse vaginal septum) in which the uterus is present and may be distended by retained blood. Integration of clinical phenotype, hormonal profile, and targeted imaging helps avoid these diagnostic errors [6,7].

Management of inguinal ovaries is driven by the risk of torsion, incarceration, and compromised ovarian reserve. Early surgical reduction and fixation (oophoropexy) is recommended, even in asymptomatic patients, to prevent ischemic injury [10]. In patients with MRKH syndrome, multidisciplinary care is essential and should include pediatric gynecology, pediatric surgery, radiology, reproductive medicine, and psychological support. Fertility counseling should emphasize that although the uterus is absent, ovarian function is usually preserved; genetic parenthood is feasible through oocyte retrieval and *in vitro* fertilization with gestational surrogacy, and uterine transplantation may be considered in select centers. Correct identification and preservation of ovarian tissue, therefore, have direct implications for long-term reproductive planning and quality of life.

This case reinforces a practical imaging message: In MRKH syndrome or suspected Mullerian agenesis, whenever ovaries are not identified in the pelvis, the inguinal canals must be actively interrogated on ultrasound and included in MRI review to exclude ectopic ovarian location.

CONCLUSION

Bilateral indirect inguinal ovarian herniation in MRKH syndrome is exceptionally rare and risks pelvic-limited imaging oversight. A structured evaluation of primary amenorrhea, including appropriate laboratory testing and meticulous ultrasound and MRI assessment of both the pelvis and inguinal canals, is essential to identify functioning ectopic ovaries, prevent misdiagnosis of ovarian agenesis, and enable timely surgical intervention to reduce the risk of torsion and preserve fertility potential.

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