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Ovotesticular disorder of sex development: bilateral ovotestes (clinical case)

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ABSTRACT

Ovotesticular disorder of sex development (true hermaphroditism) is a rare form of sex development disorder, accounting for less than 10% of all cases. This condition is characterized by the simultaneous presence of both ovarian and testicular tissue, regardless of karyotype.

This article presents a clinical case of a 61-year-old female patient diagnosed with a disorder of sex development for the first time. During a routine outpatient ultrasound examination, the ovaries appeared enlarged with active blood flow, which was disproportionate to the patient's age and postmenopausal status. The uterus and cervix were absent. The patient reported primary amenorrhea, no history of pregnancies, and an active sexual life since the age of 20. She had been married since the age of 25 and had never sought medical advice regarding infertility or amenorrhea. Further genetic and cytogenetic analysis revealed a 46,XY karyotype, associated with a high risk of gonadal malignancy. Bilateral adnexectomy was performed. Histological examination confirmed the presence of both ovarian and testicular tissue in the gonads, consistent with a diagnosis of ovotesticular disorder of sex development with bilateral ovotestes. In cases with a 46,XY karyotype, bilateral adnexectomy is indicated due to the significant risk of malignancy.

Keywords: disorder of sex development; true hermaphroditism; ovotesticular disorder of sex development; ovotestis; bilateral ovotestes; clinical case.

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Овотестикулярное нарушение формирование пола: двусторонний овотестис (клинический случай)

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АННОТАЦИЯ

Овотестикулярное нарушение формирования пола (истинный гермафродитизм) — редкая форма нарушения формирования пола, составляющая менее 10% от всех случаев. Данное состояние характеризуется одновременным наличием овариальной и тестикулярной тканей вне зависимости от кариотипа.

В статье описан клинический случай пациентки с впервые выявленным в 61 год нарушением формирования пола. На амбулаторном профилактическом приёме при проведении УЗИ обнаружено, что яичники не соответствовали возрасту и статусу менопаузы (увеличенные в размерах, активный кровоток) при отсутствии матки и шейки матки. Пациентка указала на отсутствие менструаций и беременностей, половая жизнь с 20 лет, замужем с 25 лет. По вопросам бесплодия и отсутствия менструации к гинекологу не обращалась. При дообследовании у генетиков и цитогенетическом обследовании был выявлен кариотип 46,XY, ассоциированный с повышенным риском малигнизации гонад. Проведено оперативное лечение в объёме билатеральной аднексэктомии. Гистологическое исследование выявило наличие в гонадах как овариальной, так и тестикулярной ткани, что соответствует диагнозу «овотестикулярное нарушение формирование пола с двусторонним овотестисом». При выявлении кариотипа 46,XY показана билатеральная аднексэктомия из-за высокого риска малигнизации.

Ключевые слова: нарушение формирования пола; истинный гермафродитизм; овотестикулярное нарушение формирования пола; овотестис; двусторонний овотестис; клинический случай.

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卵睾型性发育异常：双侧卵睾（临床病例）

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摘要

卵睾型性发育异常（真两性畸形）是一种罕见的性发育异常形式，约占所有性发育异常病例的不到10%。该疾病的特征是在任何核型下同时存在卵巢组织和睾丸组织。

本文报道了一例61岁女性患者的临床病例，该患者首次被确诊为性发育异常。在门诊预防性超声检查中发现，患者卵巢大小与其年龄及绝经状态不符（卵巢增大，血流丰富），但未见子宫及宫颈。患者自述自青春期以来从未有月经史，无妊娠史，自20岁起有性生活，25岁结婚。从未因不孕或月经缺失问题就诊于妇科医生。进一步的基因检测及细胞遗传学检查显示其核型为46, XY，与生殖腺恶变风险增加相关。患者接受了双侧附件切除术。组织学检查显示其生殖腺同时含有卵巢组织和睾丸组织，符合双侧卵睾型性发育异常的诊断。对于核型为46, XY的患者，由于生殖腺恶变风险较高，建议行双侧附件切除术作为预防性治疗措施。

关键词：性发育异常；真两性畸形；卵睾型性发育异常；卵睾；双侧卵睾；临床病例。

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INTRODUCTION

Disorders of sex development (DSD) are a group of congenital conditions characterized by a discrepancy between chromosomal, gonadal, and phenotypic sex. The estimated incidence is 1 in 4,500 newborns [1, 2]. DSDs include ovotesticular disorder of sex development (OT-DSD), otherwise known as true hermaphroditism, in which both male and female gonads are present concurrently. This condition is among the most uncommon forms of DSD (less than 10% of all cases), with an incidence of 1 in 100,000 newborns [3, 4]. In individuals diagnosed with OT-DSD, the karyotype 46,XX is present in 60% of cases; chromosomal mosaicism involving the Y chromosome is observed in 33% of cases, and the 46,XY configuration is identified in 7% of cases [5]. In OT-DSD, the external genitalia are usually abnormal in structure, but may appear as typically male or typically female. The primary diagnostic modality for OT-DSD is histological examination of the gonads to confirm the concurrent presence of ovarian and testicular tissues.

CASE DESCRIPTION

Patient K, female, aged 61 years, had not consulted a gynecologist and had not undergone any screening tests. For the first time, during a routine outpatient ultrasound examination at the age of 61 years, the ovaries appeared enlarged with active blood flow, disproportionate to the patient's age and postmenopausal status, in the absence of the uterus and cervix. The patient reported primary amenorrhea; no history of pregnancies, and an active sexual life since the age of 20 years. She had been married since the age of 25 years and had never sought medical advice regarding infertility or amenorrhea. Associated diseases include fibroadenomas of both mammary glands and obesity.

A physical examination revealed that the external genitalia were properly developed; the vagina was narrow; the mucosa was of normal color, and the vaginal vault was blind-ended.

According to the transvaginal ultrasound data, the uterus was not visualized; a right ovary measuring 31×44×19 mm, and a left ovary measuring 31×41×27 mm, with three single cystic small inclusions up to 5 mm and active blood flow.

According to the contrast-enhanced pelvic magnetic resonance imaging data, the uterus was not visualized; a right ovary measuring 41×24×19 mm; cystic inclusions measuring 17.14 mm, and left ovary measuring 42×38 mm, with a structure similar to the right ovary. No pelvic lymphadenopathy or abnormal foci in the pelvic bones were observed. The oncological markers were found to be within normal limits.

After a thorough review of the patient's medical history, physical examination findings, and the results of instrumental and laboratory tests, the patient was suspected of having DSD. Consequently, she was referred to a geneticist for further consultation.

Genetic counseling (Fig. 1) revealed that the proband's mother had experienced a spontaneous early miscarriage during her first pregnancy. The proband's mother had been diagnosed with ovarian cancer and died at the age of 77 years, and the proband's father had been diagnosed with lung cancer and died at the age of 65 years. According to the proband, the parents resided in a chemically polluted area. A review of the maternal family history revealed that the grandmother had type 2 diabetes mellitus and died at the age of 81 years, whereas the grandfather had arachnoiditis and died at the age of 54 years. The paternal family history was not documented.

Based on the history, clinical and genealogical analysis, clinical and phenotypic examination, and laboratory tests, the patient was given a preliminary diagnosis of testicular feminization syndrome, which should be distinguished from sex inversion syndromes of types 1–9 (Table 1).

To further clarify the diagnosis, a cytogenetic test was performed, during which 30 metaphase plates were analyzed, and the resulting karyotype was found to be 46,XY, which is indicative of a male subject.

The diagnostic techniques revealed a genetic abnormality, identified as 46,XY DSD (chromosome 9 inversion). Such patients are recommended to undergo molecular genetic testing to search for pathogenic variants in the *AR* gene, which is responsible for the development of testicular feminization syndrome, characterized by X-linked recessive inheritance. DNA testing searching for mutations in the *AR* gene is useful if the proband has female siblings, and they are planning to have children, since the patient's mother could be a carrier of a pathogenic variant in the *AR* gene. In the present study, such testing was inappropriate.

After further examination, the patient was admitted to the Clinical Hospital No. 4 of the Sechenov University (Moscow) for scheduled surgery, which included laparoscopy and bilateral adnexectomy.

Intraoperative findings included the absence of the uterus, the presence of two rudimentary, blind-ended fallopian tubes in the appendage projection, and enlarged, dense ovaries measuring up to 4 cm on both the right and left sides, with no evidence of functional activity.

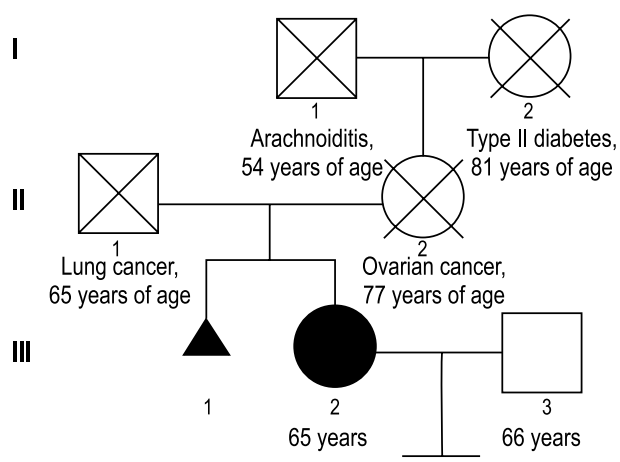


Fig. 1. Patient's Pedigree.

Table 1. Genetic spectrum of 46,XY sex reversal disorders

Condition	Gene	OMIM	Inheritance type	Key phenotypic characteristics
46,XY sex reversal, type 1	SRY	400044	Y-linked	Female phenotype, secondary sexual characteristics develop in a female pattern, uterus and fallopian tubes are normally developed or hypoplastic, dysgenetic ovaries lacking follicles Karyotype: 46,XY
46,XY sex reversal, type 2	NROB1	300018	X-linked	Female external genitalia, small or hypoplastic uterus, gonadal dysgenesis
46,XY sex reversal, type 3	NR5A1	612965	Autosomal dominant Autosomal dominant	External genitalia may be male (hypospadias, testes located in the pelvis, gonadal histology reveals fibrotic tissue without germ cells) or female (hypertrophied clitoris, small hypoplastic uterus). Low testosterone, elevated LH and FSH. Karyotype: 46,XY
46,XY sex reversal, type 4	9p24.3 deletion, candidate genes: <i>DMRT1</i> , <i>DMRT2</i>	154230	—	Short stature, trigonocephaly, flat nasal bridge, micrognathia, low-set small ears, high-arched palate. External genitalia may be male (micropenis, hypospadias, small testes) or female (hypoplastic labia majora, normal or rudimentary uterus). Karyotype: 46,XY
46,XY sex reversal, type 5	CBX2	613080	Autosomal recessive	Female external genitalia, normal uterus and cervix, normal ovaries with primordial follicles. Undetectable anti-Müllerian hormone, elevated FSH, normal LH, low testosterone, normal cortisol. Karyotype: 46,XY
46,XY sex reversal, type 6	MAP3K1	613672	Autosomal dominant	Tall stature, external genitalia may be male (hypospadias, gonadal dysgenesis) or female (enlarged clitoris, normal or hypoplastic uterus, normal fallopian tubes, hypoplastic ovaries). Sparse body hair, hirsutism. Some cases show a fully female phenotype with normal external genitalia. Karyotype: 46,XY
46,XY sex reversal, type 7	DHH	233420	Autosomal recessive	Female external genitalia, hypoplastic uterus, presence of fallopian tubes, possible epididymis, hypoplastic ovaries, risk of ovarian malignancy. Associated with muscle weakness, skin abnormalities, and sensorimotor polyneuropathy. Karyotype: 46,XY
46,XY sex reversal, type 8	AKR1C2 AKR1C4	614279	Autosomal recessive	External genitalia may be male or ambiguous. Cryptorchidism, testicular tissue capable of testosterone production. Rare cases may have rudimentary Müllerian structures. Karyotype: 46,XY
46,XY sex reversal, type 9	ZFPM2	616067	Autosomal dominant	Ambiguous genitalia, hypertrophy of the labia majora, fused labia minora, rudimentary vaginal cavity, hypoplastic uterus. Some patients may have learning disabilities, autism, and speech disorders. Karyotype: 46,XY

A histological examination of the excised appendages (see Fig. 2–9) revealed the concurrent presence of ovarian stroma and testicular tissue, consisting of tubules lined with Sertoli cells devoid of atypia and spermatogenesis, surrounded by Leydig cells. This finding suggests the presence of bilateral ovotestes. Fallopian tubes exhibited signs of hypoplasia, manifesting as a polycystic mass lined by a single layer of flattened tubal epithelium.

Based on the histological and cytogenetic data, OT-DSD (true hermaphroditism) with bilateral ovotestes was diagnosed.

DISCUSSION

OT-DSD is a rare form of DSD characterized by the simultaneous presence of both male and female gonads in an individual, regardless of karyotype. In 90% of cases, ambiguous genitalia are present at birth, whereas in the remaining cases, the external genitalia exhibit the typical structure of either a male or a female [6]. Other manifestations may include hematuria, gynecomastia, abdominal pain, primary amenorrhea, and infertility. The diagnosis of OT-DSD is typically made during the neonatal period, in cases where atypical

genitalia are observed, or in adolescence, when the primary complaint may be, for instance, the absence of menarche in patients with female-type external genitalia. The diagnosis is based on the histological examination of the gonads, which confirms the simultaneous presence of two types of tissue: ovarian and testicular.

There are several variations in the combination and position of ovarian and testicular tissues:

1. Lateral: testis and a contralateral ovary (30% of cases).
2. Bilateral: both testicular and ovarian tissues, usually represented by an ovotestis that is identified on both sides (50% of cases).

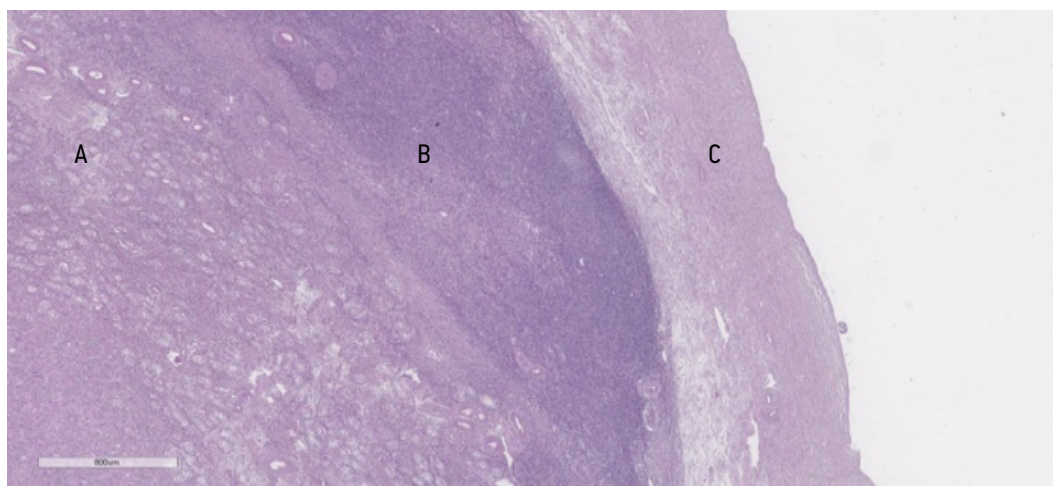


Fig. 2. Right uterine adnexa. Fragments of the ovotestis composed predominantly of testicular tissue (A) with a lobulated structure and ovarian stroma (B), enclosed within a thick tunica albuginea (C). Hematoxylin and eosin staining, $\times 800$.

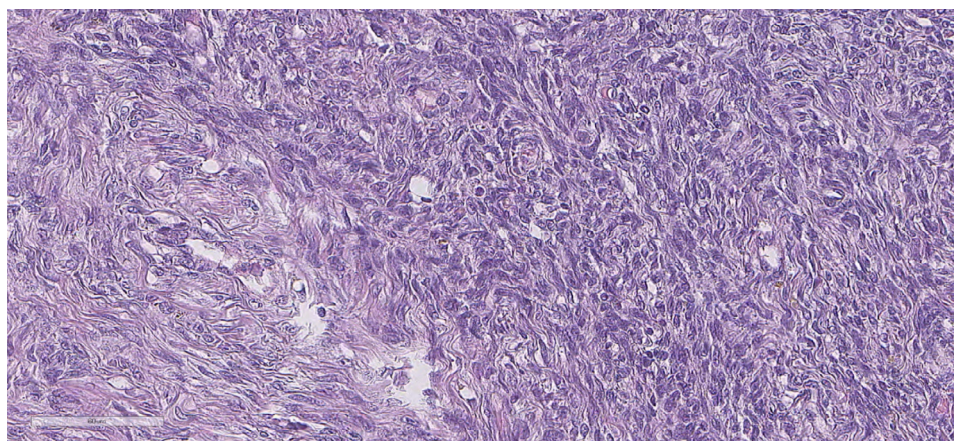


Fig. 3. Ovarian component of the ovotestis, consisting of fibroblasts. Hematoxylin and eosin staining, $\times 60$.

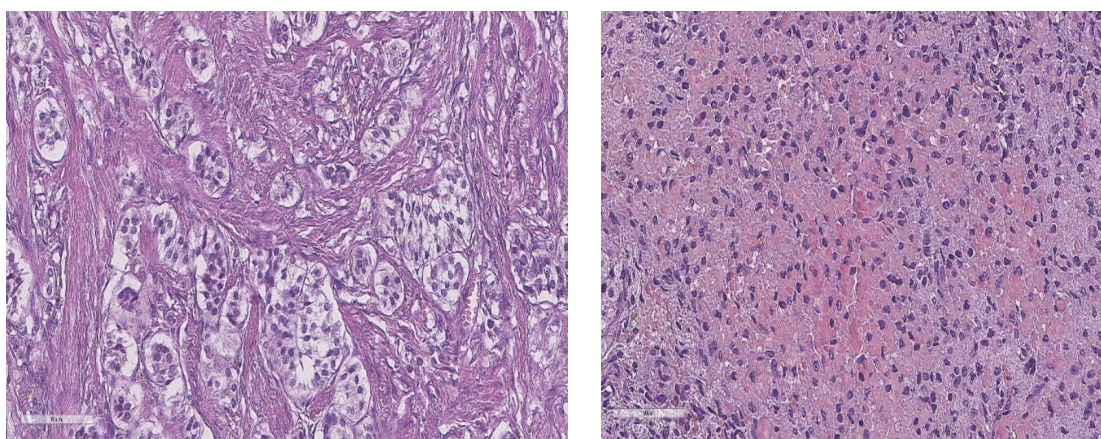


Fig. 4. Testicular component of the ovotestis, composed of seminiferous tubules lined with Sertoli cells without atypia and without spermatogenesis, along with areas of Leydig cells. Hematoxylin and eosin staining, $\times 60$.

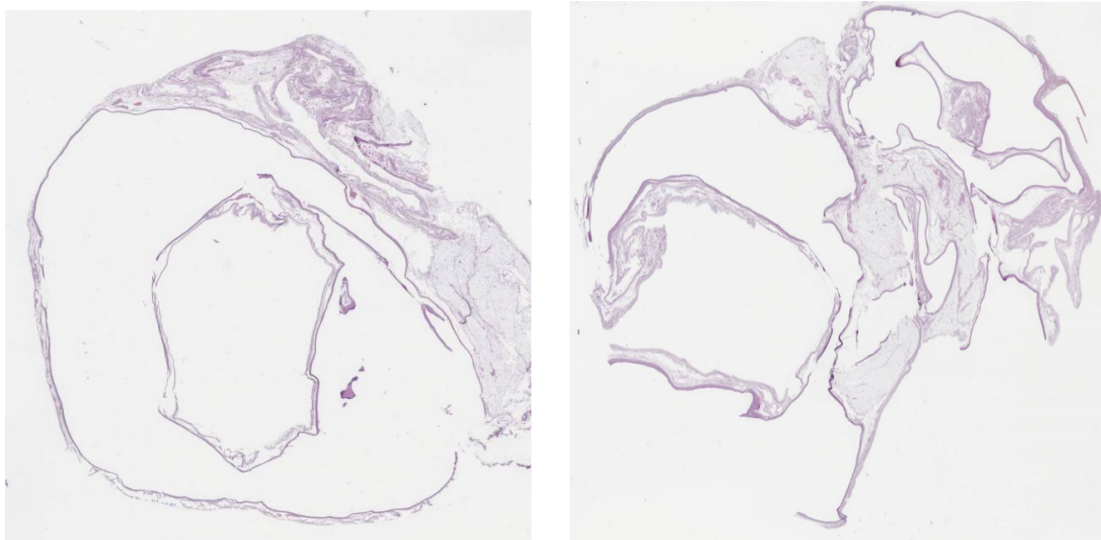


Fig. 5. Right fallopian tube with features of hypoplasia, presenting as a polycystic structure lined by a single layer of flattened tubal epithelium. Hematoxylin and eosin staining, $\times 6$.

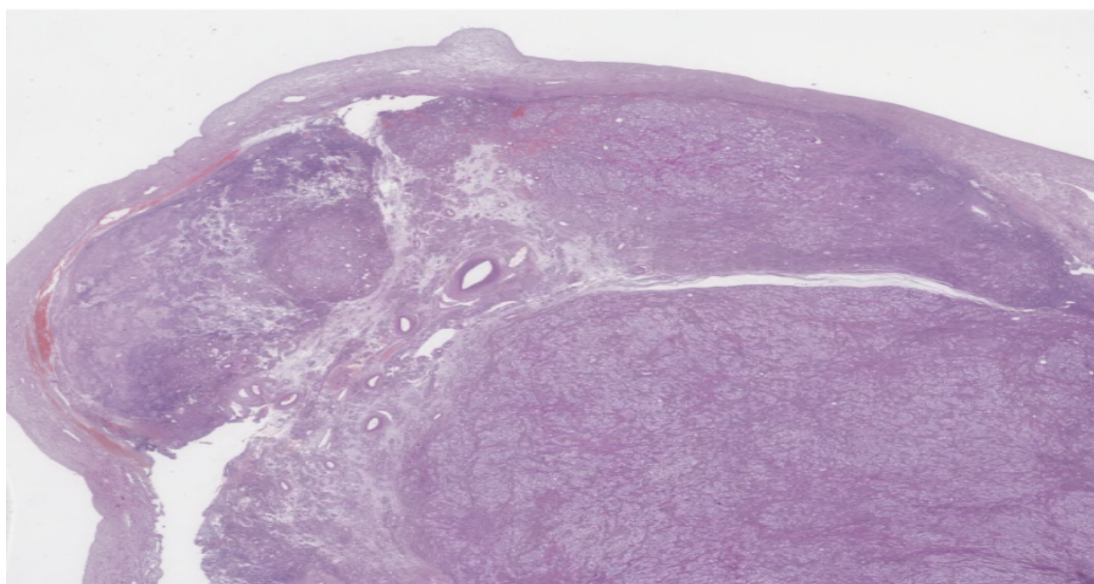


Fig. 6. Left uterine adnexa. Fragments of the ovotestis consisting predominantly of lobulated testicular tissue and ovarian stroma, enclosed within a thick tunica albuginea. Hematoxylin and eosin staining, $\times 6$.

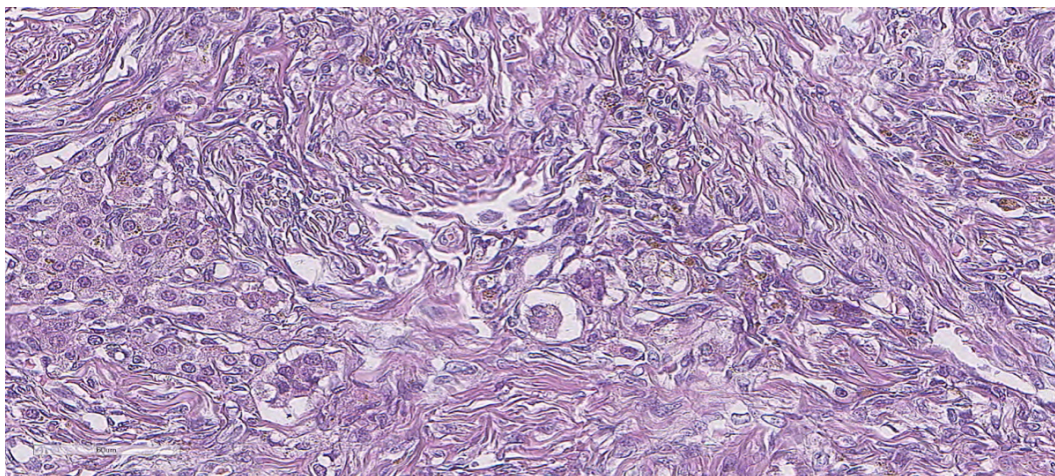


Fig. 7. Ovarian component of the ovotestis, composed of fibroblasts with clusters of Leydig cells at the border with the testicular component. Hematoxylin and eosin staining, $\times 60$.

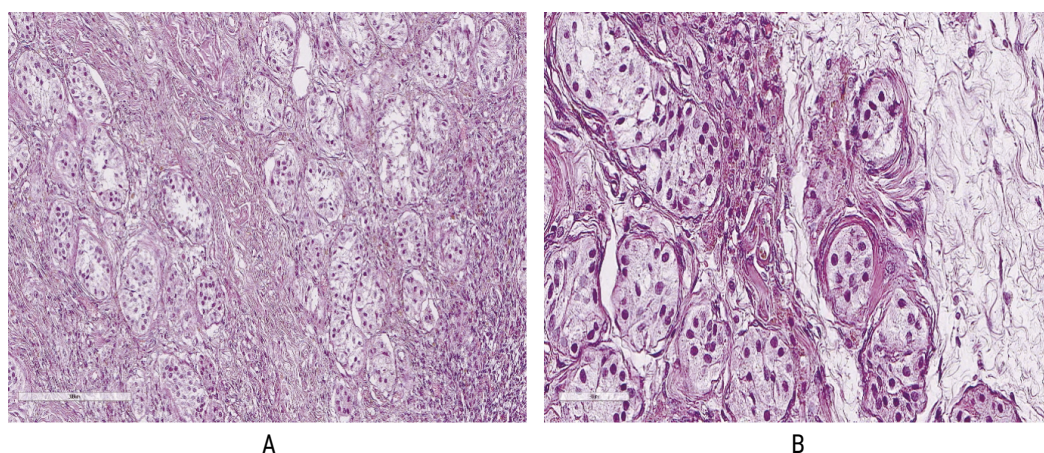


Fig. 8. Testicular component of the ovotestis, composed of seminiferous tubules lined with Sertoli cells without atypia and without spermatogenesis (A), surrounded by Leydig cells (B). Hematoxylin and eosin staining, $\times 200$, $\times 60$.

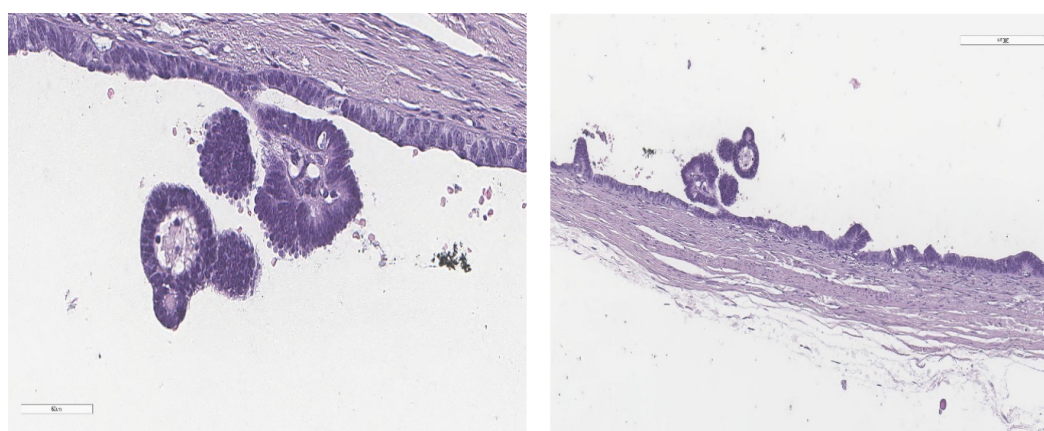


Fig. 9. Left fallopian tube with features of hypoplasia, presenting as a cystic structure lined by a single-layered tubal epithelium with occasional epithelial projections. Hematoxylin and eosin staining, $\times 200$, $\times 60$.

3. Unilateral: ovotestis on one side and a testis or ovary on the other side (20% of cases) [7, 8].

Ovotestis, a gonad consisting of ovarian and testicular tissues that externally appears as a globular structure, develops most frequently in individuals with OT-DSD [5, 9, 10]. Ganie et al. [11] conducted a comprehensive analysis of 111 cases of OT-DSD in South Africa. Based on histological studies of the interposition of ovarian and testicular tissues in ovotestis, three distinct types were distinguished (Fig. 10):

(1) mixed type, in which the outer layer consists of ovarian tissue of varying thickness; the inner layer consists of stroma with scattered foci of testicular and ovarian tissue;

(2) compartmentalized type, in which the entire upper part of the gonad consists of ovarian tissue; the lower part consists of a cluster of testicular tissue;

(3) bipolar type, in which there is a strictly polar arrangement of ovarian and testicular tissue.

The ratio of testicular to ovarian tissue in the ovotestis affects their anatomical location, with 50% of ovotestes located in the peritoneal cavity, 25% in the inguinal region, and the remaining 25% in the labioscrotal folds [12].

Testicular tissue is usually poorly developed and may be represented by immature seminiferous tubules lined with

Sertoli cells and containing spermatogonia. Leydig cells may be present. Ovarian tissue is usually more developed. Patients with OT-DSD have menstruation in 50% of cases, and pregnancies and successful deliveries have been reported [13].

In individuals with OT-DSD, a karyotype of 46,XX is determined in 60% of cases, 46,XY in 7%, and chromosomal mosaicism with Y chromosome in 33%. The presence of Y chromosome is detected mainly in patients without ovotestis. Molecular testing has shown that in 10% of cases of OT-DSD with a 46,XX karyotype, the *SRY* gene may be detected, leading to the development of male gonads. Furthermore, latent mosaicism on the Y chromosome or Y sequences in patients with a 46,XX karyotype and point mutations in the *SRY* gene in individuals with a 46,XY karyotype have been postulated as potential etiologies of abnormal gonads [14].

The presence of the Y chromosome in the karyotype of individuals with DSDs has been associated with an increased risk of gonadal malignancy and the development of germ cell tumors, including gonadoblastoma, seminoma, dysgerminoma, cystadenoma, and teratoma. Consequently, prophylactic gonadectomy is recommended for these patients [15, 16].

OT-DSD is most frequently diagnosed during the neonatal period or in early childhood, particularly when abnormal

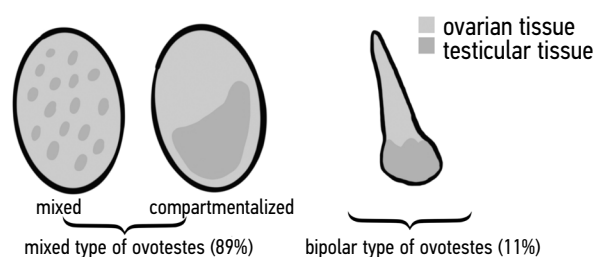


Fig. 10. Types of ovotestes.

gonads develop [17]. However, diagnosis may be delayed in some cases due to mild symptoms, poor social conditions, or limited access to well-qualified specialists in respective fields. This may lead to adverse consequences, including the development of cancer [18, 19].

A multidisciplinary approach involving gynecologists, andrologists, urologists, geneticists, psychologists, and endocrinologists is required to determine the most appropriate strategy for managing and treating patients with OT-DSD [9, 20].

CONCLUSION

The present study demonstrates a rare clinical case of OT-DSD. The diagnosis is based on histological confirmation of the simultaneous presence of ovarian and testicular tissue in an individual, regardless of karyotype. If the karyotype is 46,XY, bilateral oophorectomy is indicated due to the high risk of malignancy.

ADDITIONAL INFORMATION

Authors' contribution. N.E. Levchenko: primary contribution to the study conception, manuscript preparation, and full responsibility for all aspects of clinical case management; E.V. Sluhanchuk, F.D. Tkachenko, A.V. Rubashchenko: participation in study conception and manuscript preparation; O.A. Anurova, T.V. Filippova,

M.M. Litvinova: involvement in patient management, data collection, and case description. All authors confirm that their authorship meets the international ICMJE criteria (all authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work).

Consent for publication. Written consent was obtained from the patient for publication of relevant medical information and all accompanying images within the manuscript.

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REFERENCES | СПИСОК ЛИТЕРАТУРЫ

1. Hughes IA, Houk C, Ahmed SF, et al. Consensus statement on management of intersex disorders. *J Pediatr Urol.* 2006;2(3):148–162. doi: 10.1016/j.jpuro.2006.03.004
2. Diamond DA, Yu RN. Sexual differentiation: normal and abnormal. In: Wein AJ, Kavoussi LR, Novick AC, et al, editors. *Campbell-Walsh urology*. Philadelphia, PA: Elsevier; 2014. P. 3613–3614.
3. Blackless M, Charuvastra A, Derrtyck A, et al. How sexually dimorphic are we? Review and synthesis. *Am J Hum Biol.* 2000;12(2):151–166. doi: 10.1002/(SICI)1520-6300(200003/04)12:2<151::AID-AJHB1>3.0.CO;2-F
4. Krstić ZD, Smoljanić Z, Vukanić D, et al. True hermaphroditism: 10 years' experience. *Pediatr Surg Int.* 2000;16(8):580–583. doi: 10.1007/s003830000415
5. Krob G, Braun A, Kuhnle U. True hermaphroditism: geographical distribution, clinical findings, chromosomes and gonadal histology. *Eur J Pediatr.* 1994;153(1):2–10. doi: 10.1007/BF02000779
6. Sultan C, Paris F, Jeandel C, et al. Ambiguous genitalia in the newborn. *Semin Reprod Med.* 2002;20(3):181–188. doi: 10.1055/s-2002-35382
7. Iqbal MZ, Jam MR, Saleem M, Ahmad M. True hermaphrodite: a case report. *APSP J Case Rep.* 2011;2(2):16.
8. Hughes W, Erickson CC, Fleischmann W, Etteldorf JN. True hermaphroditism; report of a case. *J Pediatr.* 1958;52(6):662–669. doi: 10.1016/s0022-3476(58)80264-4
9. Mao Y, Chen S, Wang R, et al. Evaluation and treatment for ovotesticular disorder of sex development (OT-DSD) — experience based on a Chinese series. *BMC Urol.* 2017;17(1):21. doi: 10.1186/s12894-017-0212-8
10. Ganie Y, Aldous C, Balakrishna Y, Wiersma R. The spectrum of ovotesticular disorders of sex development in South Africa: a single-centre experience. *Horm Res Paediatr.* 2017;87(5):307–314. doi: 10.1159/000466693
11. Ganie Y, Aldous C, Balakrishna Y, Wiersma R. Disorders of sex development in children in KwaZulu-Natal Durban South Africa: 20-year experience in a tertiary centre. *J Pediatr Endocrinol Metab.* 2017;30(1):11–18. doi: 10.1515/jpem-2016-0152
12. Vilain E. The genetics of ovotesticular disorders of sex development. *Adv Exp Med Biol.* 2011;707:105–106. doi: 10.1007/978-1-4419-8002-1_22
13. Bayraktar Z. Potential autofertility in true hermaphrodites. *J Matern Fetal Neonatal Med.* 2018;31(4):542–547. doi: 10.1080/14767058.2017.1291619
14. Queipo G, Zenteno JC, Peña R, et al. Molecular analysis in true hermaphroditism: demonstration of low-level hidden mosaicism for Y-derived sequences in 46,XX cases. *Hum Genet.* 2002;111(3):278–283. doi: 10.1007/s00439-002-0772-9
15. Pleskacova J, Hersmus R, Oosterhuis JW, et al. Tumor risk in disorders of sex development. *Sex Dev.* 2010;4(4-5):259–269. doi: 10.1159/000314536
16. Abaci A, Çatli G, Berberoğlu M. Gonadal malignancy risk and prophylactic gonadectomy in disorders of sexual development. *J Pediatr Endocrinol Metab.* 2015;28(9-10):1019–1027. doi: 10.1515/jpem-2014-0522
17. Sircili MH, Denes FT, Costa EM, et al. Long-term followup of a large cohort of patients with ovotesticular disorder of sex development. *J Urol.* 2014;191(5 Suppl):1532–1536. doi: 10.1016/j.juro.2013.10.037
18. Khare M, Gupta MK, Airun A, et al. A case of true hemaphroditism presenting with dysgerminoma. *J Clin Diagn Res.* 2017;11(11):ED07–ED09. doi: 10.7860/JCDR/2017/31134/10911
19. Chen CQ, Liu Z, Lu YS, et al. True hermaphroditism with dysgerminoma: a case report. *Medicine (Baltimore).* 2020;99(22):e20472. doi: 10.1097/MD.00000000000020472
20. Meenal B, Meenakshi G, Pratibha S, et al. 46 XY ovotesticular disorder: a rare case report with review of literature. *Gynecol Minim Invasive Ther.* 2021;10(3):171–173. doi: 10.4103/GMIT.GMIT_107_19

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