Diagn Interv Radiol 2025; DOI: 10.4274/dir.2025.253612

ABDOMINAL IMAGING

ORIGINAL ARTICLE

Evaluation of the congenital absence of the vas deferens with magnetic resonance imaging: preliminary findings

Hakan İmamoğlu[1]
Erdem Sarı[1]
Özgür Karabıyık[1]
Emre Can Akınsal[2]
Gökmen Zararsız[3,4]
Oğuz Ekmekçioğlu[2]

[1]Erciyes University Faculty of Medicine, Department of Radiology, Kayseri, Türkiye

[2]Erciyes University Faculty of Medicine, Department of Urology, Kayseri, Türkiye

[3]Erciyes University Faculty of Medicine, Department of Biostatistics, Kayseri, Türkiye

[4]Erciyes University, Drug Application and Research Center (ERFARMA), Kayseri, Türkiye

PURPOSE

To date, no study provides definitive evidence for the pathogenesis of congenital absence of the vas deferens (CAVD). This study aims to evaluate the vas deferens (VD), particularly the intra-abdominal part and accompanying seminal vesicle (SV) pathologies, in search of an explanation for the pathogenesis of the disease using magnetic resonance imaging (MRI) in patients clinically diagnosed with CAVD.

METHODS

MRI scans of patients admitted to our center with clinically diagnosed unilateral CAVD (CUAVD) or bilateral CAVD (CBAVD) in the infertility clinic were retrospectively evaluated. SV hypoplasia, SV agenesis, the distal part of the VD close to the ampulla, and the intra-abdominal part of the VD were investigated. Additionally, the association of CAVD and SV pathologies was assessed.

RESULTS

Clinically and confirmed with scrotal sonography by evaluating the proximal part of the VD, 32 patients (62.7%) had CBAVD, and 19 patients (37.3) had CUAVD. In MRI, the intra-abdominal part of the VD was visible in 52.9% of all patients. The association between the intra-abdominal part of the VD and CAVD was statistically significant in the CBAVD patient group compared with the CUAVD group (Bonferroni-adjusted *P* value = 0,006). The intra-abdominal part of the VD dilatation is a new finding in CAVD and was not found in patients with CUAVD. Only 2 out of 51 patients (3.9%) had a standard SV.

CONCLUSION

In the assessment of CAVD and accompanying SV pathologies, detailed findings are obtained by MRI even in the evaluation of the intra-abdominal part of the VD. Preliminary findings in this study are consistent with the theory of acquired vasal agenesis in CBAVD.

CLINICAL SIGNIFICANCE

The detailed findings of an MRI may contribute to a better understanding of the disease.

KEYWORDS

Congenital absence of vas deferens, infertility, magnetic resonance imaging, seminal vesicle, vas deferens

Corresponding author: Emre Can Akınsal

E-mail: emreakinsal@hotmail.com

Received 19 August 2025; revision requested 15 September 2025; accepted 05 November 2025.

Epub: 19.11.2025

Publication date: xx.xx.2025

DOI: 10.4274/dir.2025.253612

Congenital absence of the vas deferens (CAVD) is one of the critical etiological causes of male infertility.[1] It is frequently observed in young and middle-aged men. Bilateral CAVD (CBAVD) has been identified in approximately 1.3% of infertile men, and unilateral CAVD (CUAVD) has been detected in 1% of cases.[2] Specifically, in cases of CBAVD, a mutation in the *cystic fibrosis transmembrane conductance regulator (CFTR)* gene associated with cystic fibrosis is frequently detected, and the majority of these cases exhibit CAVD.[3] To date, no study provides definitive evidence or a clear explanation for the mechanism of CAVD. In recent years, studies have focused on the genetic etiology of CAVD. More than 2.000 CFTR mutations have

You may cite this article as: İmamoğlu H, Sarı E, Karabıyık Ö, Akınsal EC, Zararsız G, Ekmekçioğlu O. Evaluation of the congenital absence of the vas deferens with magnetic resonance imaging: preliminary findings. *Diagn Interv Radiol*. 19 November 2025 DOI: 10.4274/dir.2025.253612 [Epub Ahead of Print].

been identified as closely associated with CBAVD.[4] Although it has been suggested that CFTR and ADGRG2 mutations are the genetic cause of the majority of CAVD cases, a precise genetic diagnosis cannot be established in 10%–20% of patients with CBAVD and 60%–70% of patients with CUAVD.[4,5] Embryologically, the seminal vesicle (SV) originates from the ampulla of the vas deferens (VD) as a diverticular structure. Due to the interrelated developmental mechanisms of the VD and SV during embryological life, SV pathologies are also commonly observed in cases where CAVD is detected.[6] The most common SV pathologies are agenesis and developmental disorders. The most frequently observed clinical finding in these patients is azoospermia; however, in cases with unilateral vasal agenesis, oligospermia, normospermia, and spontaneous fertilization are possible.[7]

Physical examination and scrotal ultrasonography can evaluate the proximal part of the VD. In cases where the etiology of infertility is being investigated in men, various radiological imaging methods are used to evaluate the intra-abdominal and distal part of the VD and SV structures, especially in cases where post-testicular pathologies leading to obstructive azoospermia are considered as a preliminary diagnosis.[8,9] Transrectal ultrasound examination is often the initial approach.[9] However, transrectal ultrasound cannot evaluate the intra-abdominal part of the VD. Moreover, the examination may cause discomfort for the patient; when patients are informed about how the transrectal ultrasound procedure will be performed, they may not accept it. In recent years, studies have been published emphasizing the importance of magnetic resonance imaging (MRI) and computed tomography (CT) in SV and VD imaging.[10,11] However, SV and VD structures in men investigated for the etiology of infertility have rarely been evaluated using CT.[6] Patients investigated for infertility are mostly young men, and the use of CT in these patients may not be appropriate due to radiation exposure. MRI does not involve exposure to ionizing radiation and has superior soft tissue resolution compared with CT.[8] It has several essential features, including its exceptional ability to detect the intra-abdominal part of the VD, higher resolution, and superior soft tissue contrast, which allow for a more detailed evaluation of SV morphology.[8,12] Despite the significant advantages of MRI in CAVD, there is a limited number of studies in the literature on the use of MRI in demonstrating CAVD and possibly associated SV pathology.[8,13,14]

This study aims to evaluate the intra-abdominal part of the VD and accompanying SV pathologies in search of an explanation for the pathogenesis of the disease using MRI in patients clinically diagnosed with CAVD.

Main points

- Magnetic resonance imaging (MRI) provides detailed findings for the assessment of congenital absence of the vas deferens (CAVD) and accompanying seminal vesicle pathologies, including the evaluation of the intra-abdominal part of the vas deferens (VD). This study identified the intra-abdominal part of the VD dilatation as a new finding.
- In the MRI of the patients with CAVD, the intra-abdominal part of the VD was observed in more than half of all patients.
- The association between the intra-abdominal part of the VD and CAVD was statistically significant in the bilateral CAVD (CBAVD) patient group compared with the unilateral CAVD (CUAVD) group. The intra-abdominal part of the VD dilatation was not found in patients with CUAVD. These preliminary findings may shed light on the pathogenesis of the CBAVD.

Methods

The Institutional Review Board approval for this single-center retrospective study was obtained from the Erciyes University Ethics Committee (decision number: 2023/171, date: March 8, 2023). Informed consent was waived for retrospective analysis.

Study population

All participants were infertile patients who applied to the infertility clinic. The study exclusion criteria were as follows: (1) prior pelvic surgery, (2) prior scrotal infection, (3) patients with uncertain diagnosis, or (4) poor image quality in the MRI. The MRI images were of patients who refused transrectal ultrasound examination to investigate the etiology of obstructive azoospermia and oligospermia. The diagnosis of CAVD was made clinically and by using scrotal ultrasonography. The MRIs of patients with clinically diagnosed CUAVD or CBAVD between January 2016 and January 2023 were retrospectively evaluated.

Magnetic resonance imaging examinations

The MRI was performed using a 1.5-T system (Signa, GE Medical Systems; Milwaukee, USA) with a 16-channel body coil, without the use of an endorectal coil and contrast material. The images were evaluated on T2-weighted sequences in axial, coronal, and sagittal planes (Table 1). CUAVD, CBAVD, SV hypoplasia, SV agenesis, the distal part of the VD close to the ampulla, and the intra-abdominal part of the VD were investigated. Normal range of the SV length and diameter was defined as 22–38 mm and 11–19 mm, respectively.[15] Normal diameter of VD in the ampulla was defined as 3–5 mm.[15] SV hypoplasia was defined as a maximum diameter of the SV being < 50% of normal or < 5 mm.[16] VD dilatation was described as a maximum diameter of the VD being > 5 mm.[15] The prevalence of these clinical conditions and the associations of CAVD and SV pathologies were examined. Imaging analysis was performed by two radiologists (H. I., O. K.) using the center's picture archiving and communication system (Sectra Workstation IDS7, Teknikringen, Sweden) in consensus. Each radiologist was blinded to the clinical diagnosis. Measurements were made in axial, coronal, and sagittal planes, and the maximum diameter and length of the SV and the maximum diameter of the VD were evaluated for the diagnosis (Figure 1).

Power analysis

A post-hoc power analysis based on the Pearson chi-squared test used to evaluate the primary hypothesis indicated a statistical power of 0.835 at an alpha level of 0.05 and

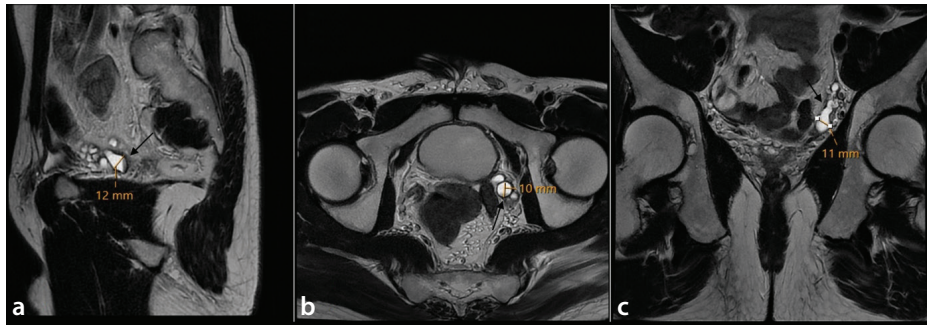

Figure 1. A 25-year-old patient with bilateral vasal agenesis and azoospermia. The T2-weighted sagittal **(a)**, axial **(b)**, and coronal **(c)** images show the intra-abdominal part of the left vas deferens dilatation (black arrows). Diagnosis was made by measuring the maximum diameter of the vas deferens.

an effect size of 0.411. Power analyses were conducted using PASS 11.0 (NCSS Inc., USA).

Statistical analysis

Statistical analyses were performed using TURCOSA (Turcosa Analytics Ltd., www.turcosa.com.tr) statistical software. Differences in MRI findings between patients with CUAVD and CBAVD and the relationship between SV pathologies and CAVD were assessed using Pearson's chi-squared test, Fisher's exact test, and age-adjusted logistic regression analysis. Data are summarized as frequencies, percentages, and corresponding confidence intervals. Confidence intervals of the estimated proportions were calculated using the Wald or Fisher approach based on the small-sample assumption (npq ≥ 5). Odds ratios were calculated using 95% confidence intervals; *P* values were adjusted using the Bonferroni approach to control for multiple testing. Adjusted *P* values of < 5% are considered statistically significant.

Results

Fifty-one patients were included in the study. The mean age of the patients was 29.53 ± 5.36 years. Clinically and confirmed with scrotal sonography by evaluating the proximal part of the VD, 32 patients (62.7%) had CBAVD, and 19 patients (37.3) had CUAVD. The clinical and radiologic characteristics of the patients are shown in Table 2. In one case, the VD was palpable at the scrotal level as a blind-ending tubular structure, whereas

Table 1. Technical data of the imaging protocol used on the 1.5-T MRI system

	T2-weighted axial imaging	T2-weighted coronal imaging	T2-weighted sagittal imaging
FOV	240	240	240
Matrix	352 × 352	352 × 352	288 × 288
TE (ms)	141	168	147
TR (ms)	3.470	6.862	3.175
Section thickness (mm)	3	3	3.5
Number of sections	23	20	20
NEX	4	4	1.5
Bandwidth (kHz)	62.5	62.5	50
Slice gap (mm)	0.5	0.5	1
Acquisition time (min)	5.28	10.06	1.37

MRI, magnetic resonance imaging; FOV: field of view, TE: echo time; TR, repetition time; NEX, number of excitations.

Table 2. Clinical and radiologic characteristics of the patients (n = 51)

Parameter	Findings
Agenesis status	
• Unilateral, n (%)	19 (37.2)—Right: 8 (42.1%), Left: 11 (57.9%)
• Bilateral, n (%)	32 (62.8)
Right testicular volume, median (min–max), mL*	18 (2–28)
Left testicular volume, median (min–max), mL*	18 (2–24)
Right epididymal morphology	
• Normal, n (%)	38 (74.6)
• Absence of the epididymal body and tail, n (%)	13 (25.4)
Left epididymal morphology	
• Normal, n (%)	36 (70.6)
• Absence of the epididymal body and tail, n (%)	15 (29.4)
Renal agenesis	
• Present, n (%)	10 (19.6)—Right: 5 (50), Left: 5 (50)
• Absent, n (%)	41 (80.3)
Semen analysis	
• Oligospermia, n (%)	4 (7.8)
• Azoospermia, n (%)	47 (92.2)
Genetic findings	
• Normal karyotype, n (%)	28 (54.9)
• Abnormal karyotype, n (%)	12 (23.5)
• Not available, n (%)	11 (21.6)

*One patient had both congenital vasal agenesis and Klinefelter syndrome, and the testicular volumes in this case were 2 mL on each side.
Epididymal and vasal structures were classified based on imaging and physical examination findings. Renal agenesis status was confirmed radiologically. Testicular volumes are presented as median and range. min, minimum; max, maximum.

in another case, it was palpable as a fibrotic band. In all other patients, the VD could not be identified at the scrotal level. All patients with oligospermia had CUAVD. Renal agenesis was detected in 5 cases in the CUAVD patient group (26.3%) and 5 cases in the CBAVD group (15.6%). In MRI, the intra-abdominal part of the VD was seen in 52.9% of all patients. The intra-abdominal part of the VD was found in 22 (68.7%) of the patients with CBAVD (Figure 2). In patients with CUAVD, the intra-abdominal part of the VD was detected in 5 patients (26.3%) (Figure 3). The association between the intra-abdominal part of the VD and CAVD was statistically significant in the CBAVD patient group compared with the CUAVD group (Bonferroni-adjusted *P* value = 0.0066) (Table 3). The CBAVD patient group had a 6.168 (1.737–21.905)-fold increased risk of an intra-abdominal part of the VD as compared with the CUAVD patient group. The intra-abdominal part of the VD dilatation was found in 5 patients (% 15.6%) with CBAVD (Figure 4), whereas no intra-abdominal part of the VD dilatation was found in patients with CUAVD (Bonferroni-adjusted *P* value = 0.2870) (Table 3).

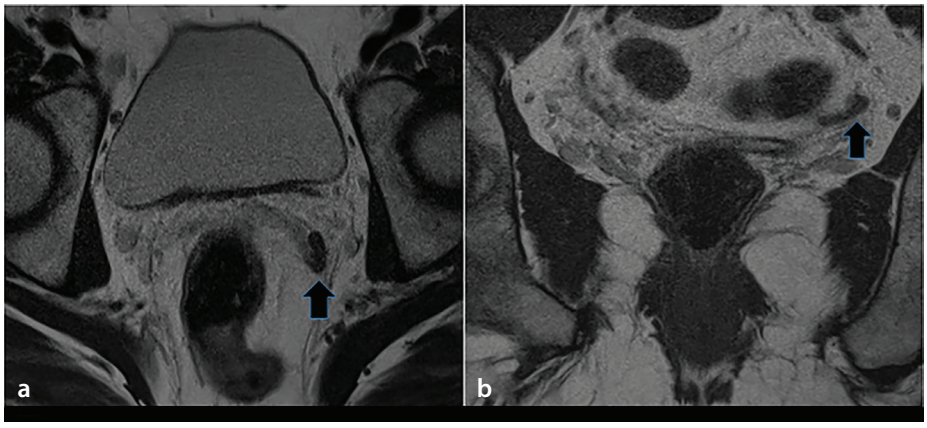

Figure 2. A 24-year-old patient with bilateral vasal agenesis and azoospermia. The T2-weighted axial **(a)** and coronal **(b)** images show the intra-abdominal part of the left vas deferens (black arrows).

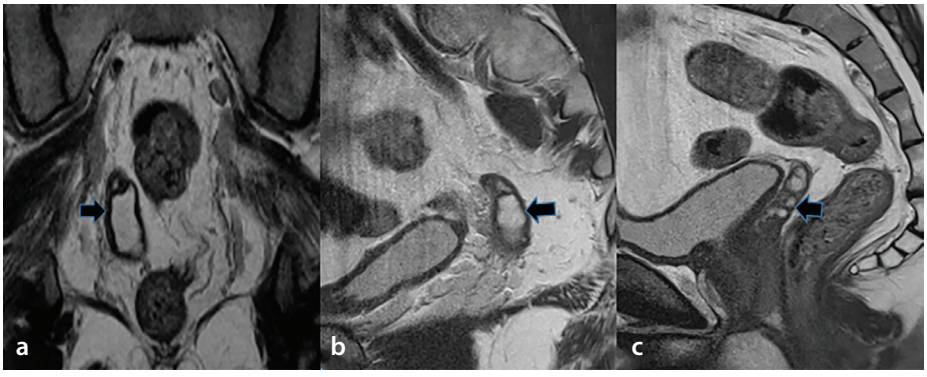

Figure 4. A 27-year-old patient with bilateral vasal agenesis and azoospermia. The T2-weighted coronal **(a)** and sagittal **(b)** images show the intra-abdominal part of the right vas deferens dilatation (black arrows). The T2-weighted sagittal image **(c)** shows a normal ipsilateral seminal vesicle (black arrow).

In 49 patients with CAVD (96.1%), accompanying SV hypoplasia and/or agenesis was detected. Among the 32 patients with CBAVD, bilateral SV agenesis was found in 15 patients (46.8%). Ipsilateral SV agenesis was found in 17 of 19 patients (89.4%) with CUAVD. Only 2 patients (3.9%) had a normal SV; 1 patient had CBAVD, and the other had CUAVD.

Discussion

In the MRI of the patients with CAVD, the intra-abdominal part of the VD was observed in more than half of all patients. Unlike the few similar studies on this subject in the literature,[13] our current study showed a higher detection rate of the intra-abdominal part of the VD in patients with CBAVD than in those with CUAVD. Furthermore, this study identified the intra-abdominal part of the VD dilatation as a new finding.

Two separate theories have been proposed for the pathogenesis of CAVD. The first theory suggests that CAVD occurs as a result of an organogenesis disorder. This theory is supported by the high prevalence of

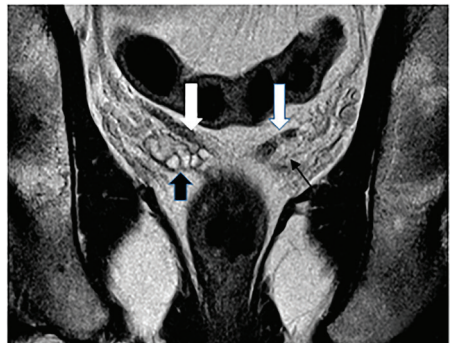

Figure 3. A 35-year-old patient with left vas deferens agenesis and azoospermia. The T2-weighted coronal image shows the bilateral distal part of the vas deferens close to the ampulla (white arrows). Although the right seminal vesicle (SV) of the same patient appears normal (thick black arrow), agenesis is present in the left SV (thin black arrow).

Table 3. The association of the intra-abdominal part of the VD and CAVD

Variables	CAVD		Bonferroni-adjusted *P* value	Age-adjusted OR (95% CI)
	CBAVD (n = 32)	CUAVD (n = 19)		
Intra-abdominal VD				
Present	22 [68.8% (95% CI: 51.3%–82.1%)]	5 [26.3% (95% CI: 11.5%–49.1%)]	0.0066†	6.168 (1.737–21.905)
Absent	10 [31.2% (95% CI: 17.8%–48.7)]	14 [73.7% (95% CI: 50.9%–88.6%)]		
Intra-abdominal VD dilatation				
Present	5 [15.6% (95% CI: 6.4%–32.2%)]	0 [0.0% (95% CI: 0.0%–17.7%)]	0.2870‡	NC
Absent	27 [84.4% (95% CI: 67.8%–93.6%)]	19 [100.0% (95% CI: 82.4%–100.0%)]		

Data values are presented as n (%). † *P* value is calculated using the Pearson chi-squared test. ‡ *P* value is calculated using Fisher's exact test. Percentages are presented with their 95% confidence intervals. Significant *P* values are shown in bold. VD, vas deferens; CAVD, congenital absence of the vas deferens; CUAVD, congenital unilateral absence of the vas deferens; CBAVD, congenital bilateral absence of the vas deferens; OR, odds ratio; CI, confidence interval; NC, not computed due to zero counts.

renal agenesis observed simultaneously in CUAVD.[4] In this study, the incidence of renal agenesis was proportionally higher in the CUAVD patient group. The second theory, more closely associated with CFTR variants, proposes that acquired vasal agenesis develops over time, with an increase in fluid viscosity and development of obstruction and degeneration in the lumen of the VD.[17] The association with cystic fibrosis in CBAVD cases is well known.[18] Conversely, literature data indicate that normal VD is detected in cases with CFTR gene expression in fetal life.[19] Additionally, experimental studies on animals without cystic fibrosis gene mutation have shown that defects occurred in the SV and/or VD secondary to obstruction and inflammation.[18-20] In the present study, the CBAVD patient group had a 6.168 (1.737–21.905)-fold increased risk of intra-abdominal part of the VD as compared with the CUAVD patient group, and although not statistically significant, a high rate of intra-abdominal part of the VD dilatation was found in CBAVD. The intra-abdominal part of the VD dilatation was not found in patients with CUAVD. We think these preliminary findings, along with some of the literature data above, may be related to the theory of acquired vasal agenesis in CBAVD.

SV pathologies (agenesis, hypoplasia) were found in 90%–100% of CAVD cases in the literature.[6,9,13,21] In the current study, this rate was 96.1%, which is consistent with the literature data. Among the 32 patients with CBAVD in this study, bilateral SV agenesis was found in 15 patients (46.8%). In the literature, the detection rate of bilateral SV agenesis in patients with CBAVD has been reported to range widely, from 9%–83%.[13,21-24] This wide range may be attributed to variations in the number of patients examined, diagnostic methods, and diagnostic criteria for SV pathologies in different studies. In the CUAVD group, SV agenesis on the same side was found in 17 out of 19 patients (89.4%). A similar rate was found in a study conducted by AbdElnaser et al.[22] (70.45% for right CUAVD–SV agenesis association and 80% for left). There is a close embryological relationship between VD and SV.[6,9,25] This process may play a significant role in the development of SV pathologies.

In this current study, only 2 patients (3.9%) had a standard (both morphology and size) SV;1 patient had CBAVD, and the other had CUAVD. Previous studies have reported a normal SV rate of 6.4%–55% in CAVD.[18,21,26,27] MRI was not used in these studies to detect SV pathologies. This may be the reason for the difference between the current study and these studies. With the increased use of MRI in patients with CAVD and accompanying SV pathologies, these rates can be determined more accurately.

The current study has some limitations, the most impactful of which is the limited number of patients. Nevertheless, the findings of this study can be confirmed and generalized through multicenter prospective studies with a large number of patients. The second limitation is selection bias from including only patients who refused transrectal ultrasound. Studies examining all patients may yield more accurate and different results. The third limitation is the retrospective design of this study, which results in a lack of genetic evaluation. Although CFTR mutation analysis was performed in a subset of our cases, the testing methods have evolved, and in some instances, patients who were initially reported as mutation-negative were later found to harbor CFTR mutations upon re-evaluation with updated techniques. Because of this inconsistency, we refrained from including these heterogeneous genetic data in the final analysis; however, we shared the genetic data in a subset of patients in Table 2. This limitation, along with the retrospective design, prevents us from definitively distinguishing congenital agenesis from acquired obstruction. Nevertheless, we included available clinical data, such as renal anomalies and semen analysis, to provide additional context. The fourth limitation is the use of 1.5-T MRI without an endorectal coil and in the absence of a standardized measurement protocol in this subject. Since there was no 3-T MRI in our hospital when patient imaging started, a 1.5-T MRI system was used. An endorectal coil was not used because patients in this study did not want uncomfortable procedures, such as a transrectal probe or an endorectal coil. A higher field strength, such as 3-T with an endorectal coil, may improve spatial resolution and provide better examination for SV and VD.

In conclusion, MRI provides valuable information on CAVD and associated SV pathologies. Our findings are consistent with—but do not prove—the theory of acquired vasal agenesis in CBAVD. Given the absence of uniform genetic testing and the evolving nature of CFTR mutation analyses, these results should be interpreted with caution. Further prospective, multicenter studies with a larger cohort, standardized genetic testing, and comprehensive clinical correlation are required to confirm these preliminary findings.

Acknowledgment

We would like to thank the Proofreading and Editing Office of the Dean for Research at Erciyes University for copyediting and proofreading service for this manuscript.

Footnotes

Conflict of interest disclosure

The authors declared that they have no conflict of interest.

References

1. Jequier AM, Ansell ID, Bullimore NJ. Congenital absence of the vasa deferentia presenting with infertility. *J Androl*. 1985;6(1):15-19. [Crossref]
2. Anguiano A, Oates RD, Amos JA, et al. Congenital bilateral absence of the vas deferens. A primarily genital form of cystic fibrosis. *JAMA*. 1992;267(13):1794-1797. [Crossref]
3. Chiang HS, Lu JF, Liu CH, Wu YN, Wu CC. CFTR (TG)m(T)n polymorphism in patients with CBAVD in a population expressing low incidence of cystic fibrosis. *Clin Genet*. 2009;76(3):282-286. [Crossref]
4. Bieth E, Hamdi SM, Mieusset R. Genetics of the congenital absence of the vas deferens. *Hum. Genet*. 2021;140(1):59-76. [Crossref]
5. Fang J, Wang X, Sun X, Cui Y, Diao F, Yang X. Congenital absence of the vas deferens with hypospadias or without hypospadias: phenotypic findings and genetic considerations. *Front. Genet*.2022;13:1035468. [Crossref]
6. Goldstein M, Schlossberg S. Men with congenital absence of the vas deferens often have seminal vesicles. *J Urol*. 1988;140(1):85-86. [Crossref]
7. Mieusset R, Bieth E, Daudin M, et al. Male partners of infertile couples with congenital unilateral absence of the vas deferens are mainly non-azoospermic. *Andrology*. 2020;8(3):645-653. [Crossref]
8. Kim B, Kawashima A, Ryu JA, Takahashi N, Hartman RP, King BF Jr. Imaging of the seminal vesicle and vas deferens. *RadioGraphics*. 2009;29(4):1105-1121. [Crossref]
9. Kuligowska E, Baker CE, Oates RD. Male infertility: role of transrectal US in diagnosis and management. *Radiology*. 1992;185(2):353-360. [Crossref]
10. Ocal O, Karaosmanoglu AD, Karcaaltıncaba M, Akata D, Ozmen M. Imaging findings of congenital anomalies of seminal vesicles. *Pol J Radiol*. 2019;84:e25-e31. [Crossref]
11. Catania R, Dasyam N, Furlan A, Borhani AA. Cross sectional imaging of seminal vesicles and vasa deferentia. *Abdom Radiol (NY)*. 2020;45(7):2049-2062. [Crossref]

12. Chen HW, Huang SC, Li YW, Chen SJ, Sheih CP. Magnetic resonance imaging of seminal vesicle cyst associated with ipsilateral urinary anomalies. *J Formos Med Assoc*. 2006;105(2):125-131. [Crossref]
13. Chiang HS, Lin YH, Wu YN, Wu CC, Liu MC, Lin CM. Advantages of magnetic resonance imaging (MRI) of the seminal vesicles and intra-abdominal vas deferens in patients with congenital absence of the vas deferens. *Urology*. 2013;82(2):345-351. [Crossref]
14. Wang H, Peng Y, Fu W, Hu X, Li C, Guan J. MRI findings of obstructive azoospermia: lesions in and out of pelvic cavity. *Abdom Radiol (NY)*. 2020;45(3):851-864. [Crossref]
15. Kim ED, Lipshultz LI, Howards SS. Male infertility. In: Gillenwater JY, Grayhack JT, Howards SS, Mitchell ME, editors. Adult and pediatric urology. 4th ed. Philadelphia (PA): Lippincott Williams & Wilkins; 2002. p. 1658–1683.
16. Raviv G, Mor Y, Levron J, et al. Role of transrectal ultrasonography in the evaluation of azoospermic men with low-volume ejaculate. *J Ultrasound Med*. 2006;25(7):825-829. [Crossref]
17. Cuppens H, Cassiman JJ. CFTR mutations and polymorphisms in male infertility. *Int J Androl*. 2004;27(5):251-256. [Crossref]
18. Mickle J, Milunsky A, Amos JA, Oates RD. Congenital unilateral absence of the vas deferens: a heterogeneous disorder with two distinct subpopulations based upon aetiology and mutational status of the cystic fibrosis gene. *Human Reprod*. 1995;10(7):1728-1735. [Crossref]
19. Gaillard DA, Carre-Pigeon F, Lallemand A. Normal vas deferens in fetuses with cystic fibrosis. *J Urol*. 1997;158(4):1549-1552. [Crossref]
20. Reynaert I, Van Der Schueren B, Degeest G, Manin M, Cuppens H, Scholte B, Cassiman JJ. Morphological changes in the vas deferens and expression of the cystic fibrosis transmembrane conductance regulator (CFTR) in control, deltaF508 and knock-out CFTR mice during postnatal life. *Mol Reprod Dev*. 2000;55(2):125-135. [Crossref]
21. Daudin M, Bieth E, Bujan L, Massat G, Pontonnier F, Mieusset R. Congenital bilateral absence of the vas deferens: clinical characteristics, biological parameters, cystic fibrosis transmembrane conductance regulator gene mutations, and implications for genetic counseling. *Fertil Steril*. 2000;74(6):1164-1174. [Crossref]
22. AbdElnaser T, Elkhiat YI, El-Azizi HM, Fatah E, Abd M, Elshibany AM, GamalEl Din SF. A cross-sectional study of the genital duct and renal anomalies in Egyptian cases of congenital absence of the vas deferens. *Hum Fertil (Camp)*. 2022;25(4):738-744. [Crossref] https://doi.org/10.1080/14647273.2021.1918352
23. Holsclaw DS, Perlmutter AD, Jockin H, Shwachman H. Genital abnormalities in male patients with cystic fibrosis. *J Urol*. 1971;106(4):568-574. [Crossref]
24. Valman HB, France NE. The vas deferens in cystic fibrosis. *Lancet*. 1969;13(2):566-567. [Crossref]
25. Kolettis PN, Sandlow JI. Clinical and genetic features of patients with congenital unilateral absence of the vas deferens. *Urology*. 2002;60(6):1073-1076. [Crossref]
26. de la Taille A, Rigot JM, et al. Correlation between genito-urinary anomalies, semen analysis and CFTR genotype in patients with congenital bilateral absence of the vas deferens. *Br J Urol*. 1998;81(4):614-619. [Crossref] Erratum in: *Br J Urol*. 1998;82(5):777.
27. Schlegel PN, Shin D, Goldstein M. Urogenital anomalies in men with congenital absence of the vas deferens. *J Urol*. 1996;155(5):1644-1648. [Crossref]