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FERTILITY DISORDERS IN PRIMARY CILIARY DYSKINESIA: MECHANISMS, DIAGNOSTICS AND MANAGEMENT OPTIONS

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ABSTRACT

Introduction and purpose: Primary ciliary dyskinesia (PCD) is a rare genetic disorder of motile cilia affecting both respiratory and reproductive systems. Because ciliary motility is essential for sperm propulsion and gamete transport in the female reproductive tract, individuals with PCD frequently present with subfertility or infertility. The purpose of this review is to synthesize current knowledge on mechanisms, clinical presentation, diagnostic pathways, and management of fertility disorders in PCD.

Description of the state of knowledge: Evidence from narrative reviews and recent cohort studies indicates that fertility impairment is more common in men than in women, with subfertility reported in up to 83% of men and 61% of women. A large cross-sectional study found infertility in 78% of men and 61% of women attempting conception and documented an increased rate of ectopic pregnancy (7.6% of pregnancies). In men, infertility is mainly due to immotile or severely dysmotile sperm resulting from axonemal defects, although many sperm remain viable; some genotypes also present with oligozoospermia, MMAF or sperm DNA damage. In women, impaired ciliary motility in the fallopian tubes and endometrium contributes to delayed embryo transport and increased ectopic pregnancy risk. Diagnosis relies on specialized PCD testing (nNO, HSVM, TEM, genetic analysis), combined with standard infertility evaluation. Assisted reproductive technologies—especially ICSI with vitality-based sperm selection—are highly effective for male PCD-related infertility, while IVF/ICSI provides favorable outcomes in women, although early pregnancy ultrasound is recommended.

Summary: Fertility disorders are a significant but manageable aspect of primary ciliary dyskinesia. A substantial proportion of affected individuals can achieve pregnancy, often with the support of ART. Early recognition of PCD in infertile patients, multidisciplinary management, and attention to genotype–phenotype correlations improve counselling and treatment planning. Further prospective studies are needed to refine prognostic predictions and optimize reproductive care in PCD.

KEYWORDS

Primary Ciliary Dyskinesia (PCD), Subfertility, Ectopic Pregnancy, Assisted Reproductive Technologies (ART), Intracytoplasmic Sperm Injection (ICSI), Sperm Motility

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1. Introduction

Primary ciliary dyskinesia is an inherited disorder characterized by abnormal or absent motility of multiple ciliated cell types, most classically respiratory epithelium, but also motile cilia and flagella in the reproductive tract [1,2]. It is usually inherited in an autosomal recessive pattern, with over 40–50 causative genes identified to date, most of them encoding axonemal or assembly proteins of motile cilia [3,4]. Clinical features typically include chronic wet cough, recurrent otitis media, rhinosinusitis and progressive bronchiectasis; approximately half of patients have situs inversus, forming Kartagener syndrome [1,5,6].

Infertility has long been recognized as a key component of the phenotype. Sperm flagella share the classic 9+2 axonemal architecture with respiratory cilia, and cilia lining the fallopian tubes and endometrium are critical for gamete and embryo transport. Defects in these structures therefore have direct consequences for human reproduction [4,7].

Despite growing interest, counselling patients with PCD about their reproductive prognosis remains difficult. Evidence is dominated by small case series and case reports, with only one recent cross-sectional cohort providing broader epidemiological data [8]. Moreover, fertility outcomes vary widely between individuals and seem to be influenced by the underlying genotype and residual ciliary function [1,8,9].

This review summarizes current knowledge on: (i) mechanisms leading to fertility disorders in PCD, (ii) diagnostic approaches to PCD-related infertility, and (iii) available management options, with a focus on ART and practical counselling of affected patients.

2. Mechanisms of fertility disorders in primary ciliary dyskinesia

2.1 Ciliary structure and function in the reproductive system

Motile cilia and sperm flagella share a highly conserved internal structure: an axoneme composed of nine peripheral microtubule doublets surrounding a central pair (9+2 arrangement), associated with outer and inner dynein arms, radial spokes and nexin links [7,9,10]. Dynein arms generate sliding forces that enable bending and coordinated beating. In the **female reproductive tract**, motile cilia are abundant along the fallopian tubes and are also found in endometrial glands. Coordinated beating towards the uterine cavity contributes to oocyte and early embryo transport, in concert with smooth muscle contractions and tubal fluid flow [1,11]. In the **male reproductive tract**, motile cilia are present in the efferent ductules, where they generate local fluid turbulence and prevent sperm agglutination, facilitating normal progression to the epididymis. The sperm flagellum is a modified motile cilium, in which axonemal and peri-axonemal structures drive progressive forward movement [1,12]. In PCD, mutations affecting dynein arms, radial spokes, central pair apparatus, docking complexes or axonemal organization lead to dysmotile or immotile cilia and flagella, explaining both respiratory and reproductive manifestations [7,9,13]**.**

2.2 Female fertility mechanisms

Subfertility in women with PCD is thought to result primarily from dysfunctional cilia in the fallopian tubes and endometrium [14]. Impaired ciliary beating may:

- slow or disrupt oocyte and early embryo transport through the tubal lumen;
- alter the distribution and clearance of tubal secretions;
- modify endometrial glandular function and early implantation environment.

These mechanisms can lead to delayed embryo transfer to the uterine cavity, increasing the risk that implantation occurs within the tube and thus predisposing to ectopic pregnancy [15]. Endometrial ciliated cells also appear to be a distinct cell population with cycle-dependent variation; dysregulation of their function has been associated with recurrent pregnancy loss in non-PCD populations, suggesting a possible contribution to early pregnancy failure in PCD as well [16].

2.3Male fertility mechanisms

Male infertility in PCD is largely driven by flagellar dysfunction, but multiple mechanisms may coexist:

- **Asthenozoospermia and immotility:**
- Many men with PCD have severely reduced motility or completely immotile sperm despite normal vitality, reflecting axonemal defects analogous to those seen in respiratory cilia [17].
- **Oligozoospermia and azoospermia:**
- Dysfunction of cilia in efferent ductules can impair sperm concentration and transport from the testis, leading to oligozoospermia or even obstructive azoospermia in some genotypes [1,4,17].
- **Structural sperm abnormalities:**
- Specific gene defects may cause combined abnormalities of motility and morphology, including multiple morphological abnormalities of the sperm flagellum (MMAF) or axonemal disorganization, further reducing fertilizing capacity [4].
- **Sperm DNA damage and centriole defects:**
- Case reports suggest that increased sperm DNA fragmentation or centriole dysfunction can be present in some PCD patients and may contribute to repeated ART failure despite ICSI [1,17,18].

Collectively, these mechanisms explain why the vast majority of men with PCD are subfertile or infertile, while a minority can still father children naturally when residual flagellar function is preserved [1,4].

2.4 Genotype–phenotype correlations

Both female and male fertility outcomes appear to depend, at least in part, on the underlying gene defect:

- In the cross-sectional cohort by Schreck et al., all women with biallelic **CCDC40** variants were infertile, whereas all women with **DNAH11** mutations conceived without fertility treatment [8].
- The narrative review by Newman et al. reports variable natural conception rates in different populations, likely reflecting the distribution of specific genotypes and associated ciliary phenotypes [1].
- Male infertility has been linked to variants in genes encoding outer dynein arm heavy chains (e.g. **DNAH5**, **DNAH9**, **DNAH11**), inner dynein arm “ruler” proteins (**CCDC39**, **CCDC40**), docking complex

proteins (**CCDC103**, **ODAD2**), central pair and radial spoke components (**HYDIN**, **RSPH1–4**), and others involved in dynein assembly or intraflagellar transport [4].

- The Novák case report highlights pathogenic variants in **SPAG1** and **DNAAF6** as causes of PCD with severe asthenozoospermia and infertility [17].

These observations emphasize the potential value of genotype-informed counselling, although current evidence is still insufficient to provide precise, gene-specific prognoses for individual patients.

3. Epidemiology and clinical spectrum of fertility disorders in PCD

3.1 Prevalence of subfertility

The true prevalence of subfertility in PCD is uncertain because of limited high-quality data and potential selection bias toward more severely affected individuals [1].

- The narrative systematic review by Newman et al. identified 108 publications reporting fertility outcomes in PCD and estimated that up to **83% of men** and **61% of women** may be subfertile in some cohorts [1].
- These estimates vary widely between studies and populations; fertile individuals are likely under-represented because they are less frequently reported [1].

The cross-sectional "Living with PCD" questionnaire provides the largest dataset to date:

- Among 168 adults who had attempted conception, **39/50 men (78%)** and **72/118 women (61%)** were classified as infertile (no clinical pregnancy after ≥12 months or need for MAR) [8].
- These findings broadly support the concept that subfertility is more prevalent in men than in women with PCD, though a substantial proportion of both sexes can conceive, particularly when supported by fertility specialists.

3.2 Natural conception and pregnancy outcomes

- The literature reviewed by Newman et al. documents numerous cases of natural conception in women and men with PCD. For example, in an earlier cohort, around 39% of women conceived within the first year of attempting pregnancy, a figure lower than the ~90% reported in the general population but clearly demonstrating that infertility is not universal [1].
- In the "Living with PCD" study, many infertile participants ultimately achieved parenthood, often after MAR, indicating that reproductive potential can be restored in a significant proportion of cases when appropriate treatment is provided [8].
- Miscarriage rates in available cohorts do not appear markedly elevated compared with the general population, although data remain sparse and heterogeneous [1].

3.3 Ectopic pregnancy

The risk of ectopic pregnancy is a particular concern in women with PCD:

- Earlier literature summarized by Newman et al. identified only four reported ectopic pregnancies in PCD, too few to estimate prevalence but suggesting a possible association with tubal ciliary dysfunction [1].
- The recent cohort by Schreck et al. provides stronger evidence: **7.6% of all pregnancies** in PCD were ectopic, and approximately **1 in 10 women** who conceived had at least one ectopic pregnancy [8].

In the general population, ectopic pregnancies account for roughly 1–2% of all pregnancies; thus, the risk in PCD appears substantially higher, although potential over-representation of complicated cases in the study sample must be considered [19-21].

These data support recommendations for early ultrasound confirmation of intrauterine pregnancy in women with PCD and rapid evaluation if pain or bleeding occurs in early gestation [8].

4. Diagnostic approach

4.1 When to suspect PCD in infertile patients

From the fertility specialist's perspective, PCD should be considered in:

- men with **persistently immotile or severely dysmotile sperm** despite normal or near-normal counts, particularly if they report a history of chronic productive cough, recurrent sinusitis, otitis media, bronchiectasis or situs inversus [4,5,22].
- women with **unexplained subfertility** or recurrent ectopic pregnancy, especially when accompanied by chronic respiratory symptoms or a family history suggestive of PCD [1,8,21].

The Novák case series illustrates typical scenarios: both men had normal or only slightly reduced sperm concentration but 0% motility on repeated samples, along with long-standing respiratory disease [17].

4.2 Diagnostic work-up of PCD

Once PCD is suspected, diagnosis should follow established international guidelines, ideally in a specialized centre. Key elements in diagnostics are:

- **Nasal nitric oxide (nNO):** markedly reduced levels are characteristic of PCD and serve as a useful screening tool [1,23-25].
- **High-speed video microscopy analysis (HSVM):** assesses ciliary beat pattern and frequency from nasal or bronchial brushings. Abnormal or absent motility supports the diagnosis [24].
- **Transmission electron microscopy (TEM):** identifies hallmark ultrastructural defects such as absence of outer and/or inner dynein arms or microtubular disorganization [9,24].
- **Genetic testing:** targeted gene panels or exome sequencing can detect pathogenic variants in a growing list of PCD-associated genes, sometimes providing genotype-phenotype correlations and allowing family counselling [4,17,24].

Combining functional and structural ciliary analyses with molecular diagnostics maximizes diagnostic yield, though not all patients have identifiable ultrastructural defects or known mutations [1,4,24].

4.3 Assessment of male fertility in PCD

Standard semen analysis according to WHO guidelines is the first-line investigation but often requires additional tests in PCD [4,17].

Key aspects include:

- **Sperm concentration and morphology:** may be normal, oligozoospermic or, in some cases, azoospermic [1,4,8]
- **Motility:** frequently shows severe asthenozoospermia or complete immotility (classical "necrozoospermia" appearance), although many sperm are still alive [17,23,24].
- **Vitality testing:** crucial when motility is absent or minimal. Techniques include hypo-osmotic swelling test and laser-assisted immotile sperm selection (LAISS) to identify viable sperm for ICSI [4,26].
- **Further investigations:** in selected cases, sperm DNA fragmentation assays (e.g. TUNEL), assessment of mitochondrial function or electron microscopy may help explain repeated ART failure and guide prognosis [17].

4.4 Assessment of female fertility in PCD

Women with PCD should undergo standard infertility work-up, tailored to their age and clinical history:

- evaluation of ovulatory function and ovarian reserve;
- imaging of the uterus and adnexa;
- assessment of tubal patency (e.g. hysterosalpingography or sonohysterography), recognizing that tubal motility, not just patency, may be impaired;
- review of previous pregnancies, miscarriages and ectopic pregnancies [1,8].

Because tubal ciliary dysfunction rather than mechanical obstruction is the main problem, some women may have normal imaging but still experience delayed embryo transport or recurrent ectopic pregnancy [1,8].

5. Management options

5.1 General principles and counselling

Management of fertility disorders in PCD should be multidisciplinary, involving respiratory specialists, fertility specialists, geneticists and, when needed, psychologists [1].

Key counselling points include:

- Subfertility is **common but not universal**; some individuals conceive spontaneously [8,14,27].
- Fertility prognosis may vary by genotype, but current data are insufficient for definitive gene-specific predictions [4,8].
- Assisted reproduction is often successful, especially with appropriate laboratory techniques, but treatment may be more complex and prolonged than in idiopathic infertility [1,28,29].
- Women should be informed about the increased risk of ectopic pregnancy and the need for early pregnancy monitoring [8].

Preconception optimization of respiratory health (e.g. airway clearance, infection control, smoking cessation) is also recommended to minimize maternal morbidity during pregnancy [1].

5.2 Natural conception and expectant management

Given that a significant minority of patients, particularly women, can conceive naturally, expectant management may be reasonable in younger couples with favourable prognostic features (e.g. some sperm motility, normal ovarian reserve and no prior ectopic pregnancy) [1].

However, prolonged delays should be avoided, especially in older women or when semen analysis shows severe abnormalities. A lower threshold for early referral to fertility services is appropriate in PCD compared with the general population [1,8].

5.3 Assisted reproductive technologies in women with PCD

Evidence on ART outcomes in women with PCD is limited but encouraging:

- The narrative review by Newman et al. describes multiple successful pregnancies after IVF or ICSI in women with PCD, including those with previous ectopic pregnancies or prolonged infertility [1].
- In the Schreck cohort, 59 infertile women underwent MAR, and **69%** achieved pregnancy with its help [8].

Practical considerations:

- Standard ovarian stimulation and IVF protocols can usually be used; there is no consistent evidence that PCD alters ovarian response or embryo quality [13,16,30].
- Because of the higher ectopic pregnancy rate, early ultrasound (around 5–6 weeks gestation) is recommended after both spontaneous and ART pregnancies to confirm intrauterine implantation [8].
- Single-embryo transfer may be preferred to reduce obstetric risk in women with significant respiratory compromise [1].

5.4 Management of male infertility: ICSI and beyond

ART is central in male PCD-related infertility, particularly ICSI:

- Intracytoplasmic sperm injection bypasses the need for sperm motility and allows the use of immotile but viable spermatozoa [13,16,30,31].
- When ejaculated sperm are absent or extremely scarce, surgical retrieval (e.g. testicular sperm extraction, TESE) provides an alternative source for ICSI [23,31,32].

Laboratory techniques highlighted in the male infertility review and case report include:

- **Vitality-based sperm selection** using hypo-osmotic swelling test or LAISS to identify live sperm among immotile cells, improving fertilization rates [4,17].
- Thorough semen work-up, including **sperm DNA fragmentation** testing in couples with repeated ART failures, to detect additional factors limiting success [17].

While many case series report good fertilization and live birth rates with ICSI in PCD, there are also examples where repeated cycles fail, possibly because of underlying DNA damage or centriole abnormalities [1,4,17]. These couples require realistic counselling and careful weighing of further ART versus alternative options such as donor sperm.

5.5 Ectopic pregnancy risk and obstetric care

Given the increased ectopic pregnancy rate specific measures are advisable in women with PCD:

- **Early serum hCG and transvaginal ultrasound** in every pregnancy, whether natural or after ART, to confirm intrauterine location [8].
- Rapid evaluation of abdominal pain or bleeding in early pregnancy.
- Close collaboration between fertility specialists and obstetricians familiar with PCD, particularly in women with significant lung disease [1,8].

Miscarriage rates do not appear markedly elevated, but data are limited, and routine obstetric care should include standard surveillance for fetal growth and maternal respiratory status [1].

5.6 Future perspectives

Emerging research in PCD, particularly in gene and RNA-based therapies, offers theoretical hope for disease-modifying treatments that might ultimately improve fertility [4]. Approaches under investigation include:

- viral vector-mediated gene replacement,
- CRISPR-based gene editing,
- transcript-level interventions such as mRNA or antisense therapies.

However, these strategies face major challenges, including large gene sizes, efficient delivery to airway and reproductive tissues, and safety concerns, and none are ready for clinical application to infertility at present [33,34].

In the nearer term, better genotype-phenotype correlation studies and prospective registries are needed to refine counselling on natural fertility potential, ART success rates and obstetric risks in PCD [1,8].

6. Conclusions

Primary ciliary dyskinesia is a rare but important cause of subfertility and infertility in both women and men. Fertility impairment results from dysfunctional motile cilia in the fallopian tubes, endometrium and efferent ductules, as well as intrinsic sperm flagellar defects, often compounded by genotype-specific structural abnormalities [7-18].

Available data suggest that infertility is more frequent in men than in women, but many individuals of both sexes can conceive, especially with appropriate fertility support [1,8]. The recent cross-sectional cohort highlights a high prevalence of infertility and an increased risk of ectopic pregnancy, reinforcing the need to address fertility systematically in routine PCD care and to ensure early pregnancy monitoring [15,19,20].

From a clinical perspective, PCD should be considered in infertile men with immotile sperm and respiratory symptoms, and in women with unexplained subfertility or recurrent ectopic pregnancies [17]. Diagnosis relies on specialized ciliary and genetic testing, complemented by standard infertility investigations.

Management is centred on individualized use of ART. ICSI with ejaculated or testicular sperm, guided by vitality-based selection techniques, is the cornerstone of treatment for male PCD-related infertility, while IVF/ICSI offers good prospects for women, provided ectopic pregnancy risk is actively managed [1,4,30-34].

Further prospective and genotype-informed studies are needed to define optimal diagnostic pathways and treatment strategies, but current evidence already supports proactive, multidisciplinary fertility counselling for all adolescents and adults living with PCD.

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