

Transient Complete Asthenozoospermia

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ABSTRACT

Limited research exists on the mechanisms underlying asthenozoospermia associated with acquired ciliary dyskinesia. Primary ciliary dyskinesia links respiratory pathology with infertility and provides a basis for a potential mechanism. The aetiology of asthenozoospermia is often unclear and may be secondary to direct or indirect effects on sperm motility. Here, we report a case – with a brief clinical review – of recovering sperm motility after diagnosis of complete asthenozoospermia coinciding with resolution of chronic respiratory infections. The patient is a 36-year-old male, with initial semen analysis demonstrating 100% immotile sperm. Following the resolution of chronic respiratory infection, subsequent analysis demonstrated functional improvement with 76 million sperm/mL, 8% progressive motility and 4% strict morphology. Our case reinforces a potentially underappreciated role of environmental risk factors in infertility, with a focus on the patient's history of infections and other risk factors for acquired ciliary dyskinesia, which should be kept in mind when treating patients with asthenozoospermia.

KEYWORDS: *Asthenozoospermia, infertility, respiratory infections*

INTRODUCTION

Infertility is a concern that affects 10%–20% of couples worldwide, with up to 50% of cases having a significant male factor.^[1] Complete asthenozoospermia or 100% immotile spermatozoa, is a relatively uncommon form of male factor infertility with poor fertility prognosis, impacting 1 in 5000 men.^[2] Causes of complete asthenozoospermia are divided into two categories: necrozoospermia and ultrastructural defects in sperm flagellum. Necrozoospermia may be secondary to a diverse set of causes, including oxidative stress, environmental pollutant exposure or prolonged anejaculation.^[2] While literature highlights a range of causes, the aetiology of each case is often unknown.

Regarding ultrastructural defects, previous case reports associate sperm immotility and chronic upper and lower respiratory infections. This association is especially evident amongst patients with primary ciliary dyskinesia.^[3] Acquired ciliary dysfunction, secondary to chronic infections, smoking and

other factors, has similarly been associated with infertility^[4,5] – emphasising the impact of environment supplementary to genetics.

Despite evidence supporting the association between respiratory infections and sperm immotility, the temporality and mechanism of the association are unclear. Here, we propose the idea that extragonadal pathology causing ciliary dysfunction may lead to structural alterations in sperm. We present a case of an adult male with transient complete asthenozoospermia in the setting of recurrent respiratory infections.

CASE REPORT

The patient was a 36-year-old male referred for a possible male factor in a secondary infertile relationship. His past medical history included Crohn's disease, obesity status post-gastric bypass, tobacco use (2 pack years), asthma and environmental allergies. A semen analysis in 2015 demonstrated low volume without mention of

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abnormal motility. He and his wife had attempted to achieve a pregnancy for 24 months with intercourse every 2–3 days. The couple had a prior conception, with one son born in 2019. His physical examination revealed bilateral grade 1 varicoceles. Semen analysis at the initial visit, shown in Table 1, demonstrated low volume (1.3 mL) and 100% immotile sperm.

On further evaluation, he noted recurrent respiratory infections attributed to household mould during home renovations, as well as known diagnoses of asthma and allergic rhinitis, with evidence of chronic eosinophilia (>9.0%). He was recommended for a repeat semen analysis following infectious treatment and avoidance of mould exposure.

At his 3-month follow-up visit, the patient denied additional respiratory symptoms given mould removal from his home. His follow-up complete semen analysis demonstrated asthenozoospermia with 0.8 mL of volume, a concentration of 76 million sperm/mL, 8% progressive motility and 4% strict morphology. Following that visit, the patient provided written informed consent for publication of his case. The couple has continued to pursue timed intercourse for natural conception. No positive pregnancy had been confirmed at 4 months following the updated semen analysis.

DISCUSSION

Of the factors contributing to male infertility, asthenozoospermia encompasses a significant

proportion.^[6] Complete asthenozoospermia, while rarer, is a particular challenge. Its treatment requires assisted reproductive technologies due to the necessity of rapidly progressive motile sperm to penetrate cervical mucus.^[7] In understanding the cause and treatment of complete asthenozoospermia, an interplay of environment and risk factors must be considered. Our review is limited to phenotypic asthenozoospermia seen in our patient. We recognise this limitation in data regarding polymorphisms associated with infertility, such as those seen in endothelial nitric oxide synthase.

As this case suggests, there may exist a previously unidentified relationship between chronic respiratory infections and asthenozoospermia. The improvement in sperm motility following the resolution of respiratory symptoms indicates a mechanism for transient disruption of sperm function that may be remediated by removing the offending agents. As mentioned previously, primary ciliary dyskinesia is a genetic syndrome characterised by chronic lung disease, respiratory infections and infertility due to the shared aetiology of ciliary dysfunction.^[3] In contrast, secondary or acquired ciliary dyskinesia is commonly observed within the respiratory tract and is attributed to damage from factors such as smoking, asthma, allergic rhinitis and other lung pathologies.^[4] Our patient's risk factors for developing acquired ciliary dyskinesia include chronic fungal infections, a history of smoking, asthma and environmental allergies.

Table 1: Results of patient's semen analyses

	Baseline semen analysis (simple count and motility)	Semen analysis at 3-month follow-up (complete)
Liquefaction (complete)	Complete	Complete
Appearance (grey/opalescent)	Grey/opalescent	Grey/opalescent
Volume (≥ 1.5 mL)	1.3	0.8
Viscosity index (0)	0	0
Concentration ($\geq 15 \times 10^6$ mL)	79	76
Total sperm number ($\geq 39 \times 10^6$)	102.7	60.8
Motility grade 0 (immotile %)	100	87
Motility grade 1 (slow, nonprogressive) %		5
Motility grade 2 (slow, progressive) %		6
Motility grade 3 (rapid, progressive) %		2
Progressive motility ($\geq 32\%$)		8
Total motile sperm ($\geq 12 \times 10^6$)		4.9
Strict morphology ($\geq 4\%$)		4
Total motile normal ($\geq 1 \times 10^6$)		0.19
Peroxidase-positive leucocytes ($< 1 \times 10^6$ /mL)		<1
Round cells ($< 5 \times 10^6$ /mL)		<1
Agglutination (-/+)		+
Narrative	Diagnosis: No motility was observed. Viability unknown. The patient did not state any issue at the time of collection	Diagnosis: Asthenozoospermia. Patient did not state partial loss of the sample at the time of collection

Currently, it is unclear if the pathophysiology of asthenozoospermia associated with acquired ciliary dyskinesia shares similar mechanisms to that of primary ciliary dyskinesia. It is possible that extragonadal ciliary dysfunction may lead to alterations in sperm motility. There is limited research on this matter, and it is unclear if decreased sperm motility is due to direct effects on ciliary function or indirect effects from recurring infections and a chronic inflammatory state. Several studies have linked chronic infection, inflammation and oxidative stress with changes in sperm function and male infertility, including one by Bonanno *et al.* that demonstrated evidence of direct damage to sperm by reactive oxygen species in patients with asthenozoospermia.^[8,9] For a subset of patients, complete asthenozoospermia may have its root cause in a congenital anomaly with variable penetrance that structurally impairs sperm flagellum. Alternatively, it may arise from an acquired source, such that exposure to chronic respiratory infections or environmental allergens in those already predisposed to poor ciliary motility leads to subsequent loss of function.

The temporality and permanence of this condition also require further study. One case report demonstrated transient complete asthenozoospermia following COVID-19 infection and shares similarities with our patient.^[10] In this case, the patient previously had a normal semen analysis but presented with 0% motility 2 weeks after the onset of COVID-19 infection. Three months after his initial diagnosis, his motility returned to 44% without intervention. Given these similarities, it is worth exploring the possibility that transient asthenozoospermia could lead to misdiagnosis of permanent male factor infertility in men who may later regain fertility.

In the case of our patient, while several risk factors could have contributed to the immotility, we hypothesise the complete asthenozoospermia was likely driven by his respiratory pathology and allergen exposure, supported by his improvement in progressive motility once environmental exposure was removed and his symptoms resolved.

The mechanisms underlying ciliary dysfunction are not fully elucidated. Structural defects or epithelial metaplasia are implicated in the respiratory tract, and we propose that these alterations may present systemically. It remains unclear whether these are transient changes in sperm flagella or the result of damage from systemic inflammation.

CONCLUSION

The current case highlights the importance of continued follow-up and repeat semen analysis after the removal of possible insult. In addition, a thorough history of previous respiratory infections and environmental exposure should be obtained. Future investigation

should focus on identifying patients at risk for transient asthenozoospermia and the evolution of its pathology.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Author's contribution

All authors were involved in the writing and editing of the manuscript. The senior author was responsible for the conceptualisation of the work.

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Conflicts of interest

There are no conflicts of interest.

Data availability statement

Data will be available from corresponding author upon reasonable request.

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