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# Transient Azoospermia Caused by a Febrile Episode Secondary to Meningitis



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## Abstract

Fever of greater than 39 degrees C for over two days can lead to poor semen quality and azoospermia. During spermatogenesis the spermatocytes and spermatids are sensitive to thermal extremes. We report a case of secondary infertility that was associated with azoospermia secondary to an acute febrile episode linked to meningitis. Spontaneous recovery of sperm count five months after the febrile event allowed for a natural pregnancy after ovulation induction with clomiphene citrate in our patient. A full history of any febrile episodes in the previous three months should be a part of the work-up for azoospermia or poor semen parameters in all patients. In cases where a febrile episode was noted, a repeat semen analysis in three months may demonstrate normal parameters and can avoid unnecessary and expensive testing and treatment.

**Keywords:** Azoospermia; Abnormal semen analysis; Male infertility; Fever

## Introduction

In humans the male gonad requires a specialized temperature environment to produce sperm. Spermatogenesis is optimal at a temperature of approximately one degree below normal body temperature. There are at least three known causes of male infertility that have been related to excessive heat including cryptorchidism, varicocele and fever. The undescended testes in cryptorchidism expose the testis to suprascrotal temperature resulting in abnormal sperm development. Varicoceles are thought to cause interruptions in the countercurrent heat exchange system causing increased scrotal temperature resulting in infertility through altered sperm production [1]. Febrile illnesses increase scrotal temperature and have been reported as an important cause of reversible male infertility [2].

We describe a case of azoospermia associated with a severe febrile episode caused by viral meningitis that spontaneously resolved and resulted in adequate sperm production to conceive naturally.

## Case Presentation

A 36 year-old G1P0010 female and 39 year-old male were referred for consultation concerning secondary infertility for two years. Her evaluation included a hysterosalpingogram with a normal uterine cavity and patent fallopian tubes, a low

mid-luteal progesterone of 3.7ng/ml with irregular cycles indicating anovulation, an FSH of 6.4mIU/ml and an anti-mullerian hormone level of 1.74ng/ml on cycle day 3 suggesting normal ovarian function. Based on this evaluation the patient was counseled about her options and she decided to attempt ovulation induction with clomiphene citrate coupled with timed intercourse or intrauterine insemination (IUI) depending on the results of her partner's semen analysis.

A normal semen analysis demonstrated 3 cc, 61 million sperm per cc, with 49% rapid progressive motility, and 12% normal forms by Krueger strict morphology. Prior to cycle initiation, the patient cancelled her IUI cycle secondary to her husband being ill and admitted to the hospital. One month later, her husband was diagnosed with viral meningitis at which time he had a fever of 39.5 degrees Centigrade for 48 hours.

After her husband's recovery, they decided to initiate her IUI cycle but repeat semen analysis two months later showed 2 cc volume but no sperm seen in the raw or concentrated specimen (Table 1). The IUI cycle was postponed with plans for repeat semen analysis in two months which revealed 14.9 million sperm per cc with abnormal parameters (Table 1). Ovulation induction with clomiphene citrate coupled with IUI was prescribed.

**Table 1:** Semen Analysis Parameters in Relation to Febrile Episode.

Time Related to Febrile Episode	Volume (2-5cc)	Count (20 to 200 Million /Cc)	Motility (>40% Rapidly Progressive)	Normal Forms (>14% Strict Morphology)
- 1 month	3.0	61	49	14
Fever 39.5C				
+2 months	2.0	0	NA	NA
+4 months	2.0	14.9	27	3
+5 months	2.0	73	79	13

One month later, or five months from the original febrile episode, semen analysis showed 3 cc, 73 million sperm per cc, with 79% motility, and 13% normal forms. The patient called the clinic later that month reporting a positive home pregnancy test on a clomiphene citrate cycle. She delivered a term infant via cesarean delivery secondary to breech presentation.

**Discussion**

**Table 2:** Classification of Azoospermia.

Obstructive (Post-Testicular)	
	Surgical
	Vasectomy
	Hernia repair with scarring
	Genetic/Congenital
	J15deferens (cystic fibrosis carrier)
	Infectious
	Prostatitis, epididymitis, orchitis
Non-obstructive Pre-Testicular	
	Kallmann Syndrome
	Hypothalamic and/or Pituitary dysfunction
	Drug induced (anabolic steroids, testosterone)
Non-Obstructive Testicular	
	Klinefelter's Syndrome
	Sertoli cell-only syndrome
	Cryptorchidism (undescended tested)
	Radiation and/or chemotherapy
	Mumps orchitis
	Varicocele
	Spermatogenic arrest
	Y chromosome microdeletions
	Systemic illness (Cancer, febrile episode)

and this three lines place above the table heat effect on spermatogenesis. Available evidence suggest that the magnitude of spermatozoa damage after exposure to heat is determined by

the location of spermatozoa in excretory system, susceptibility to the effect of heat and the degree and period of heat exposure. Less mature spermatozoa, in the terminal portion of the seminiferous tubules and in the head of the epididymis, are more susceptible to heat than mature spermatozoa in the tail of the epididymis and the vas deferens [2].

The causes of azoospermia can be divided in two broad categories (Table 2). Obstructive azoospermia (OA) occurs when normal sperm are being produced in the testes but a blockage in the vas-deferens does not allow the sperm to be ejaculated. The most common causes of OA can be genetic, congenital or acquired. These include infections (epididymitis, orchitis, prostatitis and venereal diseases), vasectomy, congenital absence of the vas deferens associated with cystic fibrosis, and complications of surgery such as hernia repair [3].

Non obstructive azoospermia (NOA) can be caused by abnormalities within the testicle, deficiencies in the reproductive hormones produces by the hypothalamic/pituitary glands, genetic, congenital or acquired causes. Genetic causes include Klinefelter syndrome and deletions in the Y-chromosome. Other causes of NOA include hypospermatogenesis, maturation arrest, Sertoli-only syndrome, Germ cell aplasia, testicular cancer, and medical treatments such as chemotherapy or radiation therapy. When the clinical history reveals a febrile episode and no other causes, such as in this patient with prior fertility and with a recent episode of spinal meningitis, it is reasonable to wait for a few months and repeat the semen analysis before initiating expensive testing.

Studies on paraplegic men have provided convincing evidence linking environmental heat to reduced sperm motility. A significantly higher mean testicular temperature was noted in paraplegic men during the daytime than in normal men who had been sitting still for at least 20 minutes. Studies of sperm motility in the paraplegic men revealed an association between high scrotal temperature and lack of motile sperm [2]. Damage to human sperm chromatin structure has been identified in the case of a febrile episode related to influenza [4]. It was determined that increased histone/protamine ratios had latent effects on sperm chromatin structure and the transient production of abnormal sperm.

A large study of semen of 122 infertile men with testicular hyperthermia either due to occupational exposure to high temperature or varicocele, revealed that 106 men had azoospermia and 14 men had oligozoospermia. All of the oligospermic men showed a high percentage of sperm with abnormal morphology and impaired motility. This study reiterates that high intratesticular temperature causes partial or complete spermatogenic arrest and may lead to increased production of morphologically abnormal sperm with impaired motility [5].

This case demonstrates that temporary azoospermia can be caused by elevated body temperatures. We report viral meningitis and fever as a cause of acute and temporary azoospermia, with recovery of sperm parameters and fertility four months later. The evaluation of men who present with azoospermia should include a history of any febrile episode within the past four months. In

the absence of other obvious etiologies, it is reasonable to repeat a semen analysis in four months before initiating further testing and treatment.

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