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Androlog Summary

The Clinical Conundrum of Complete Asthenospermia

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Men with complete asthenospermia pose intriguing problems to the male reproductive clinician, and questions regarding this condition arise on *Androlog* from time to time. In 1998, Dr Malpani asked:

> For a man with 100% immotile sperm in the ejaculate, of which 10% are live, are pregnancy rates better with intracytoplasmic sperm injection (ICSI) (after selecting live sperm with a hypo-osmotic swelling test), or is it better to offer TESE-ICSI (ICSI with testicular sperm extraction)?

In 2000, Andy Meacham submitted the following queries:

> I am currently evaluating a 28-year-old gentleman whose semen analyses have shown excellent sperm counts on 2 occasions (99 and 102 million per mil, respectively) but 0% motility. He is completely normal on physical examination and has no correctable factors that I can identify. His past medical history is noncontributory. We have not thus far performed any additional tests to evaluate sperm viability. He and his wife are very interested in establishing a pregnancy and are willing to consider ICSI if this would be of help. I would be interested in the group's current thoughts on 2 questions: 1) How optimistic are you that hypo-osmotic swelling (or other techniques) would reliably identify sperm suitable for ICSI in a case such as this? 2) Does anyone feel any optimism that testis biopsy might yield motile sperm that could then be used in an ICSI procedure? Your thoughts on this would be much appreciated.

Recently, Jay Sandlow posed the following questions:

> I know we have covered this numerous times, but I would like an opinion (regarding a case). The (case) is a patient with immotile sperm. (I hesitate to call it "necrospermia" because not all of the sperm are dead.) This is a healthy patient (who is adopted, so we do not know family history), with no gonadotoxin exposure. On physical exam, he has normal-sized testes and a small to moderate left varicocele. His semen analyses have shown

normal volumes (3–4.5 mL), low counts (5–15 million/mL), no motile sperm with 50%–60% viability, and 0% strict morphology. Cystic fibrosis transmembrane conductance regulator (CFTR) and poly-T testing are negative, as is an abdominal ultrasound. (I have seen 2 patients with polycystic kidney disease present this way.) He is reluctant to undergo a transrectal ultrasound to rule out ejaculatory ductal obstruction unless I really think this is likely. My question is, does he need any further evaluation, or can we just use testicular sperm (which will have a higher viability to his ejaculated sample)? What are the other potential etiologies? Should I send his sperm/testis tissue for electron microscopy?

Grace Centola proposes the possibility of immotile cilial syndrome, as well as decay of motility in the semen analysis procedure itself:

> Have you considered "immotile cilia syndrome?" Does this patient present with frequent upper respiratory infections, pneumonia, etc, that would suggest immotile cilia on cells other than in the reproductive tract? A nasal or bronchial biopsy would confirm. Years ago I reported on such a patient, whose diagnosis was confirmed with electron microscopy of both nasal/bronchial biopsy and sperm. At that time, ICSI was not an option, so the patients conceived with donor sperm. One other possibility: make sure that the semen was collected properly—we have all seen scenarios of improper collection and transport/storage that resulted in all nonmotile sperm. An example, was the semen stored in a refrigerator prior to delivery to the lab, or collected in a latex condom? Just some suggestions.

Marc Goldstein raises the concern of anti-sperm antibodies and notes the unlikelihood of ejaculatory ductal obstruction. Like Dr Centola, Dr Goldstein recommends electron microscopic evaluation for immotile cilial syndrome:

> I would get an anti-sperm antibody assay. If it were highly positive, that would indicate obstruction and suggest that motile sperm could be found in the proximal epididymis or efferent ducts or testis. With a volume of 3–4.5 mL, ejaculatory ductal obstruction is exceedingly unlikely. I would do electron microscopy to look for absent dyneine side arms characteristic of Kartageners. ICSI can work in these men. I would also treat the semen with Pentoxifylline to see if any sperm twitch, thus obviating the need for any invasive methods of sperm acquisition.

Larry Lipshultz finds that ejaculatory ductal obstruction may very well be present:

> I am concerned as to why this man is "reluctant to undergo a transrectal ultrasound to rule out ejaculatory ductal obstruction." It would be very likely to see a partial obstruction with either a stenosis or a cyst in a patient with no motility and low counts in the face of normal testicular size. Seminal vesicle aspiration can also be done at the same time, and indeed you might find motile sperm. Consequently, you might be able to convert a patient who is now destined to only in vitro fertilization/ICSI to one who might be able to initiate a spontaneous pregnancy. If the transrectal ultrasound is negative (and I would do the seminal vesicle aspiration regardless of what you see), thencertainly electron microscopy of the sperm tail would be indicated to look for microskeleton defects.

In an opposing view, Dana Ohl posits that ejaculatory ductal obstruction and anti-sperm antibodies would be unlikely etiologic agents:

I find it hard to believe that this result is due to cytotoxic antibodies or partial ejaculatory ductal obstruction, as the reason for total lack of motility in either case should be that the sperm are dead. Noncytotoxic antibodies rarely will cause total lack of motility.

I would think electron microscopy of the sperm tail would be more likely to find the cause. To Grace's comments, although nasal swab usually will be positive because of common genetics of the sperm flagellum and nasal epithelium, and patients with defects will have pulmonary symptoms. However, this is not always the case. We had a patient with totally normal nasal epithelium on two occasions, with total absence of the inner and outer dyneine arms in the sperm, also on two occasions.

The comment about two patients with polycystic kidney disease is interesting. Please see: H Okada et al (Assisted reproduction for infertile patients with 9+0 immotile spermatozoa associated with autosomal dominant polycystic kidney disease. *Hum Reprod.* 1999;14:110–113).

Some may find it disquieting that a clinical condition that has reared its perplexing head in several *Androlog* queries over the years continues to elicit many different views from established authorities with great experience in the field of male reproductive medicine. Yet such varied discourse is in fact exhilarating: it reminds us that we still practice in a young field with many unanswered questions and that we look forward to many discoveries in reproductive nature yet to come.

Footnotes

Note: Postings to *Androlog* have been lightly edited before publication.