The Parasite that Wasn’t:  
A Case of Detached Ciliary Tufts in Cerebrospinal Fluid

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Case Presentation
A 16-year-old girl with congenital hydrocephalus presented to her neurosurgeon with symptoms of ventriculoperitoneal shunt malfunction. Over the previous few days, the patient had become increasingly nauseous and lethargic, and her headache had worsened. On fundoscopic examination, mild papilledema was observed, and retinal venous pulsations were diminished. Her blood pressure was elevated. Cognitive function was intact. Based on history, neurological findings, and a relatively non-compressible catheter valve, imaging of the shunt apparatus and a shunt tap were indicated.

Plain film revealed an intact distal catheter, while CT scan ruled out proximal disconnection. CT scan further revealed a lateral ventricle hydrocephalus, though it was not until shunt tap was performed that the presumed obstruction was localized. A shunt tap, in which CSF is collected from a valve reservoir that sits subcutaneously and posteriorly to the auricle, revealed elevated opening pressure, indicative of an obstruction distal to the valve. A sample of CSF was taken for biochemistry, Gram stain, and culture.

All biochemical parameters, including CSF protein and glucose concentrations, were normal. Gram stain was negative and a white cell differential uncovered nothing unusual. However, upon microscopic examination of the CSF sample, several unicellular, ciliated “swimming things” were evident in each low-power field. On average, cells measured 16 mm in diameter and had circumferentially arranged cilia. A second shunt tap was performed and a second CSF sample obtained for further microscopy. Again, these parasites, with which the laboratory technicians had no familiarity, were observed. Thus, a parasitologist was called in to identify these organisms. What is the differential diagnosis for “swimming things” in cerebrospinal fluid?

Presumptive Diagnosis: Opportunistic Protozoal Infection
There are many organisms, both protozoan and helminthic, that can parasitize the central nervous system. Only a select few, however, fulfill enough of the aforementioned criteria (i.e., unicellularity, ciliary-based motility) to be considered in this case. The first, a ubiquitous ameboflagellate by the name of Naegleria fowleri, causes a fulminant disease called primary amebic meningoencephalitis (PAM) in an extremely small percentage of people who come in contact with it. As this protozoan tends to inhabit warm, fresh water, most victims acquire the organism while swimming.1 Amebae enter the swimmer’s nose and access the subarachnoid space of the CNS via the olfactory neuroepithelium and olfactory nerves.1 Although a typical incubation period is one to two weeks, once fever and headache are present, a patient has only 72 hours before coma and death ensue.12 Thus, the possibility of antemortem diagnosis is negligible, with only three survivors reported worldwide.1 In the parenchymal tissue of the brain, the organism takes on an ameboid feeding form; however, in CSF Naegleria exists in its flagellated form, and can therefore appear to “swim”. As the patient in this case has no recent history of swimming, and a presentation inconsistent with PAM, it is unlikely that she is suffering an infection with N. fowleri. In addition, PAM typically mimics bacterial meningitis in terms of CSF findings – polymorphonuclear pleocytosis, elevated protein, and very low glucose – none of which were present in this case.

Unlike Naegleria fowleri, trichomonads are not generally associated with CNS infection, although there is evidence to suggest that they should be considered when faced with an unidentified ciliated/flagellated protozoan, regardless of the site of infection. Three trichomonads – Trichomonas tenax, Trichomonas vaginalis, and Pentatrichomonas hominis – have all been reported in association with or as the primary cause of disease in humans; although typically, only T. vaginalis is thought to be pathogenic. Trichomonas tenax, normally a commensal inhabitant of the mouth, can cause infection of the respiratory tract in people with underlying pulmonary disease.3 Similarly, P. hominis, an enteric trichomonad, is more likely to be recovered from individuals with symptomatic bowel disease.3 Trichomonas vaginalis can cause sexually transmitted trichomoniasis in humans, and is actually the most common parasitic cause of STD.4 Although trichomonads are known to be site- and, to a certain extent, host-specific,3 cases of cross-species infection and recovery of trichomonads from aberrant locations have been reported. For example, in 1997, a 34-year-old immunocompromised man died of acute meningoencephalitis secondary to infection with Tritrichomonas foetus,5 an organism that usually infects the GU system of cattle. How this flagellate was able to survive in the nervous system of a human host remains unexplained, though the patient’s immunocompromised state was a likely facilitating

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factor. Severe meningoencephalitis and ventriculitis were evident at autopsy, and histopathological examination of the meninges revealed many trichomonads, confirmed by SEM to be *T. foetus*. As with a *Naegleria* infection, CSF showed a marked pleocytosis.5

The case of *T. foetus*-meningoencephalitis bears particular relevance to the present case as it demonstrates the ability of trichomonads to proliferate in CSF, and induce an inflammatory response that could lead to complications in a shunted individual. Although trichomonad infection should be considered in the differential for the patient reported herein, it is unlikely that the “swimming” organisms isolated were members of this flagellate order. On high-power light microscopic examination, it is very clear that trichomonads lack the circumferential arrangement of flagella noted in this case. In terms of presentation, there was nothing in the patient’s history to suggest infection with *T. vaginalis*, or a state of immunosuppression. In addition, her lack of fever, pleocytosis, and elevated CSF protein would argue against protozoal infection.

Single-cellular kinetoplastid flagellates, in particular, the CNS-invading trypanosomes that cause African sleeping sickness, can appear to “swim” in CSF, hence their inclusion in this discussion. Members of the *Trypanosoma brucei* complex can be distinguished from the organisms in this case because of their long, singular polarized flagellum that appears to whip, not beat. Furthermore, transmission of these trypanosomes can only occur through the bite of a tsetse fly vector, endemic to Africa. Although the initial presentation of African sleeping sickness may include headache, nausea, and lethargy, inconsistent parameters that could lead to complications in a shunted individual. Although trichomonad infection should be considered in the differential for the patient reported herein, it is unlikely that the “swimming” organisms isolated were members of this flagellate order. On high-power light microscopic examination, it is very clear that trichomonads lack the circumferential arrangement of flagella noted in this case. In terms of presentation, there was nothing in the patient’s history to suggest infection with *T. vaginalis*, or a state of immunosuppression. In addition, her lack of fever, pleocytosis, and elevated CSF protein would argue against protozoal infection.

**Pathological Discussion**

The second CSF sample was used to prepare wet mounts for Nomarski interference contrast (NIC) microscopy. Both live and formalin-fixed organisms were photographed and measured using an ocular micrometer. As previously mentioned, cells averaged 16 µm in diameter, and had cilia arranged around their periphery (Figure 1). Careful microscopic examination of cells revealed an absence of nuclei, and conventional ciliate culture medium inoculated with CSF failed to grow these ciliates.

To summarize, the “swimming things” found in this patient’s CSF were devoid of nuclei and failed to grow in vitro. Although their size was in the range of both *Naegleria* and trichomonads, they did not resemble any ciliated or flagellated protozoan known to the parasitologist. Coupled with a normal biochemical profile of CSF and the process of elimination, these unique morphological data led the team to a new differential diagnosis: detached ciliary tufts of unknown origin.

**Detached Ciliary Tufts**

Ciliated epithelia are common throughout the respiratory and genital tracts of humans. Ciliated cells also line the tympani, Eustachian tubes, and ventricles of the brain. At any of these sites, ciliated cells have the potential to detach by constricting and severs their luminal portions, leaving behind their nucleus and basal cytoplasm.6 Thus, anucleate apical fragments of ciliated cells can be a non-pathologic finding in many body fluids.6 Although the nucleus is generally retained basally, mitochondria are pinched off with the apical cytoplasm, allowing the cell fragment to remain motile for several days.7 The vigorous ciliary motion that detached ciliary tufts (DCTs) can exhibit in wet mount, creates a diagnostic dilemma of sorts as it emulates that of ciliated protozoa.5 As evidenced by this case and others,6,10 DCTs can be easily confused with members of the protozoa.

DCTs are found most frequently in nasal secretions, sputum, and cervicovaginal smears;6 they are also a common finding in bronchoalveolar lavage.11 Although the formation of DCTs has been unequivocally linked to respiratory viral infection, it has yet to be demonstrated that DCTs have pathologic significance in any other circumstance.6 The term ciliovorticella (CCP) has been used to describe DCTs that occur in respiratory tract specimens, and because of the established pathologic connotation, the term should only be applied in this context.6 Unlike CCP, DCTs in cervicovaginal smears and peritoneal dialysates of women result from ciliated oviduct epithelium undergoing a normal, cyclic exfoliation12 (Figure 2). Thus, their presence in these fluids should be construed not as a pathologic finding, but a physiological one.6,12 Not surprisingly, post-menopausal women and men undergoing continuous ambulatory peritoneal dialysis (CAPD) are not likely to have DCTs in their effluent.8 To our knowledge there are no prior reports of DCTs found in CSF. To recap, the finding in body fluids of ciliated cell fragments up to 15 µm in diameter and lacking internal structure, should be diagnostic for DCTs.6 Parasitic infection can be further excluded by an inability to culture these “ciliates” in vitro.

![Figure 1. Nomarski interference contrast micrograph of a ciliated cell fragment isolated from the CSF of a young patient. Note the circumferential arrangement of cilia (arrows). Bar = 16 µm.](image1)

![Figure 2. Detached ciliary tuft from a Papanicolaou-stained smear. Photograph reproduced with permission from Atlas of Human Parasitology, 4th ed. (Ash and Orihel, eds), p. 371.](image2)
As the patient reported herein has a CSF shunt, there are two potential portals through which DCTs could have accessed the catheter reservoir: the ventricular end and/or the peritoneal end of the catheter. In this particular shunt apparatus, the proximal catheter passes from a cerebral ventricle (typically lateral) to the valve reservoir via a cranial burr hole. From the valve, the distal portion of the shunt catheter continues along a subcutaneous tunnel until it terminates in the peritoneal cavity. Given this shunt anatomy, there are also two potential sources of DCTs in this patient. First, the DCTs isolated from her shunt reservoir could have arisen from the cerebral ependyma. Alternatively, DCTs of oviduct origin could have entered the peritoneal cavity and accessed the shunt reservoir through retrograde migration. Compelling arguments can be made to support either of these possibilities.

Support for Ependymal Origin of DCTs
Numerous investigations into the causes of proximal shunt malfunction have consistently demonstrated choroid plexus and ependyma to be common sources of obstructive agents. In fact, choroid plexus appears to be the most common cause of ventricular catheter obstruction reported. In their examination of 91 ventricular catheter ends, Sekhar et al. (1982) found choroid plexus to be the obstructive material in 35 catheters, and ependyma in 15. The prevailing notion is that if proximal catheter inlets are in contact with choroid plexus and/or ependyma (after normalization of ICP and ventricular size), then the tissue can grow into and eventually occlude the catheter lumen. That only a few of the 40-50 small catheter inlets need be patent to maintain adequate CSF flow suggests that ependymal tissue could be present in the shunt catheter without causing obstruction. In light of ependymal morphology (Figure 3), it is conceivable that cell fragments could detach and, because of proximity, easily enter the catheter lumen, hence their discovery in the shunt reservoir. Of particular interest to this case is the finding of choroid plexus, along with other embolic materials, as a cause of distal catheter obstruction (i.e., peritoneal end). That cells of the ventricular lining can be found so far distally substantiates their ability (“intentional” or otherwise) to enter the shunt apparatus, migrate along a flow gradient, and potentially cause obstruction. Although this scenario is plausible, that DCTs are merely cell fragments lacking certain identity cues, make it difficult to localize their site of origin. Therefore, the possibility that her DCTs were from another anatomic locale must be considered.

Support for Oviduct Origin of DCTs
In terms of morphology, the DCTs isolated from this patient very closely resemble those known to be oviduct epithelial cells (compare Figures 1 and 2). Unfortunately, as this is the first reported case to intimate ependymal DCTs, we are lacking a comparison photograph. Clearly, the literature supports an oviduct origin and in this patient, there is a very reasonable explanation of how oviduct DCTs could be present in CSF. Since 1986, DCTs have been found in the peritoneal washings of 25 women, most of whom were of reproductive age. In most of the case reports, DCTs were a surprising, incidental finding. Although there exists no evidence that ciliated cells shed into the peritoneal cavity elicit an inflammatory response, in a shunted individual such as this patient, one must be cognizant that oviduct DCTs can be formed with each menstrual cycle, and can thus make their way into the shunt catheter lumen. Whether or not they cause obstruction in motile form, or can withstand the shunt fluid dynamics remains to be determined.

Patient Outcome
After the first two shunt taps, the patient’s blockage appeared to resolve spontaneously without neurosurgical intervention. Her ICP normalized and symptoms regressed. She was discharged home and monitored over the next several weeks by her family physician, with no recurrence of symptoms. It was not determined if the DCTs found in the patient’s CSF were actually causing the obstruction that led to her admission. Commonly, distal obstruction is attributed to shunt contact with omentum. Despite the possibility that DCTs may have been an incidental finding in this case, it raises the issue of awareness. Clearly, laboratory technicians and other medical personnel should be familiar with the occurrence of DCTs, particularly in the context of respiratory infections. General awareness of DCTs could not only circumvent misdiagnosis and needless pharmacotherapeutic regimes, it could also prevent utilization of costly resources such as infectious disease consultations. Without this familiarity, the presence of DCTs could easily be mistaken for disease with underlying infectious or inflammatory etiology.
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