

Introduction

In July 2008, I met Manuel in a retinal clinic in the outpatients department of Sydney Eye Hospital. I observed a clinical consultation between a retinal fellow, Manuel and his Mother. In the brief encounter in that darkened consultation room, I wondered what sort of pathology would bring a seemingly healthy young man to this place. The retinal fellow disclosed to me that this young man has toxoplasmosis in his right eye. Shortly after, I set out to discover, what is toxoplasmosis, and what is Manuel's story? Owing to the brevity of that encounter, most of this case study is based on information obtained from Manuel's medical records.

Social History

There is little information in the medical record about Manuel's social history. What little that is known, is taken from medical admissions, progress notes and the nursing care plan. Manuel is 26 years old. Born in Venezuela, I am unable to determine what age he migrated to Australia with his family. Currently, he is employed in a bank and still lives with his family. He lists English as his preferred language, although I did observe him speaking Spanish with his mother.

Visual History

During the clinic consultation, Manuel stated that he had been diagnosed with optic neuritis as a teenager, and that he attended the Sydney Eye Hospital for his condition. I was unable to confirm this diagnosis, as the appropriate medical records have been destroyed. Manuel wears glasses for myopia. His prescription is unknown.

Relevant Medical History

There is no evidence of any relevant medical or surgical history in his medical record.

Epidemiology

Epidemiology is the study of the distribution and determinants of diseases within human populations (Greenberg et al 2005). It is at this point that special distinction must be made between toxoplasmosis and ocular toxoplasmosis. Toxoplasmosis is the term used to describe the clinical manifestations of someone infected by the micro-organism *Toxoplasma gondii* (*T. gondii*). Ocular toxoplasmosis is the term used to describe the clinical manifestations of someone whose eye/s are infected by *T. gondii*. Toxoplasmosis does not always manifest as ocular toxoplasmosis. However, ocular toxoplasmosis is the most widely observed cause for posterior uveitis and retinochoroiditis seen in immunocompetent human beings (Lightman & Towler 1998: 67).

The incidence of toxoplasmosis varies greatly throughout the world. There are vast differences between continents, nations, and even amongst local communities. There are great worldwide disparities between the number of people with toxoplasmosis who go on to develop ocular toxoplasmosis. For example, France and Sao Paulo (Brazil) demonstrate

similar infection rates of *T. gondii*, but ocular toxoplasmosis is far more common in Sao Paulo (Muccioli et al 2007).

The statistics are both conflicting and compelling. According to estimates, total worldwide incidence of *T. gondii* infection is between 13 - 15% (Koo et al 2006), or up to one third of the world's population (Muccioli et al 2007). The incidence of *T. gondii* infection in the United Kingdom has been estimated at 16 - 40% (Hill et al 2002). In the United States, 3 - 70% of the population are infected with *T. gondii* (Levinson & Rikkers 2004). In continental Europe, Central and South America, incidence of *T. gondii* infection is 50 - 80% (Hill et al 2002). About 30 - 40% of Australia's population are infected with *T. gondii* (Dickenson 2001).

More specifically, the incidence of symptomatic toxoplasmic retinochoroiditis in the United Kingdom has been estimated at 0.4 cases/100 000 population/year. There may be 1.26 million people with ocular toxoplasmosis living in the United States (Koo et al 2006). In one study conducted at Sydney Eye Hospital, ocular toxoplasmosis accounted for 20% of posterior uveitis in 245 patients seen over 5 years (Wakefield et al 1986).

Pathophysiology

Toxoplasmosis and ocular toxoplasmosis are caused by the protozoa *Toxoplasma Gondii* (Lightman & Towler 1998: 67) Protozoa are unicellular eukaryotes (Solberg 2004). The life cycle of *T. gondii* is complex, and was only fully understood in 1970 (Lightman & Towler 1998: 67). There are three distinct phases, eggs (oocysts), active parasites (tachyzoites) and cysts containing inactive parasites (bradyzoites) (Koo et al 2006). Infection is normally acquired by ingesting *T. gondii* eggs found in cat faeces, or food or water contaminated by cat faeces (Muccioli et al 2007). Infection may also be acquired by ingesting undercooked meat that contains *T. gondii* cysts (Levinson & Rikkers 2004). Finally, infection may also occur in utero, as *T. gondii* can be transmitted by vertical means. *T. gondii* active parasites pass via the placenta from Mother to foetus (Hill et al 2002).

In the case of acquired infection, eggs transform into active parasites. Active parasites course around the body taking up residence in the central nervous system, eyes, skeleton and myocardium. Natural immune responses transform the active parasites into cysts. Cysts may remain dormant for years, but can also reactivate and transform into active parasites again (Koo et al 2006). Systemic clinical manifestations of acquired toxoplasmosis include lymphadenopathy, sore throat, fever, malaise, night sweats, myalgias and maculopapular rash (Muccioli et al 2007). Clinical manifestations of congenital toxoplasmosis are dependant on the timing of transmission. Generally speaking, the earlier during the pregnancy that foetal infection occurs, the more severe the congenital effects, which range from retinochoroiditis, hydrocephalus, convulsions, intracerebral calcification and death. The most common clinical manifestation of congenital infection is ocular toxoplasmosis (Hill et al 2002).

Ocular toxoplasmosis was first observed in the eye of an infant in 1923. However, focal retinochoroiditis in adults was presumed to be the result of tuberculosis or syphilis. The discovery of *T. gondii* cysts in enucleated eyes changed this thinking (Hay & Dutton 1995). Most clinical presentations of ocular toxoplasmosis are a subsequent reactivation of congenital *T. gondii* cysts contracted in utero (Ng & McCluskey 2002). However, there is growing evidence to suggest that ocular toxoplasmosis may also be caused by acquired *T. gondii* infection (Levinson & Rikkers 2004). Inactive parasites residing in retinal cysts, reactivate into active parasites, or newly acquired active parasites take up residence in the retina. Active parasite activity initiates an inflammatory response in the retina, which destroys retinal tissue (Koo et al 2006). It must be stressed that the choroid is usually involved as well, thus leading to the diagnosis of toxoplasmic retinochoroiditis. Choroidal involvement allows ocular toxoplasmosis to be further classified as a type of posterior uveitis (Lightman & Towler 1998: 69).

When retinal tissue destruction occurs, there are several visible phenomena. Retinal lesions whether they be congenital or acquired have the same appearance. Lesions in their active state appear as white and fluffy, sometimes described as hazy retinochoroiditis. This lesion is often described as a 'headlight in the fog' (Muccioli et al 2007). Inactive lesions appear as white and well defined with pigmented margins (Ng & McCluskey 2002). Some significance is placed on the area where lesions occur. For instance, congenital ocular toxoplasmosis is believed more likely to cause lesions bilaterally, and involve the macula. Acquired ocular toxoplasmosis is believed to cause lesions unilaterally and involve the peripheral retina (Muccioli et al 2007). The cycle of inflammatory responses cause lesions to scarify the retina. Retinochoroidal scars are observed in about 80% of people who contracted ocular toxoplasmosis in utero (Levinson & Rikkers 2004).

Client's Presenting Clinical Manifestations

Manuel presented to the emergency department of Sydney Eye Hospital on Christmas Day 2007. Manuel stated that he developed a 'floater' on the 24th December, and that presently he has blurred vision in his right eye. The triage nurse noted that there was red colour desaturation in the right eye, and no pain. Nursing staff tested his visual acuity with a Topcon machine while he wore his glasses. His visual acuity scores were RVA 6/9-1 and LVA 6/4.5-1. There was no improvement with pin holes in the right eye.

Differential Diagnosis

The differential diagnosis begun the moment Manuel presented to the emergency department. It is apparent that he has decreased visual acuity in his right eye. More importantly, vision was not improved with the use of pin holes. Pin holes allow light to be focused more directly on the macula in instances of refractive error (Chang 2008). Manuel

has a refractive error that is corrected by glasses, so it is likely that his decreased vision is not a result of further refractive deterioration.

The medical officer checks for red reflex and noted that the red reflex was darker in the right eye compared with the left. The clinical significance of the red reflex test is to determine if there is anything preventing the retina from appearing red (normal) when light is shone through the pupil. Since there are numerous media that the light needs to pass through to reach the retina, such as the lens and vitreous, it is unclear at this stage what could be preventing the red reflex from normal appearance.

Manuel is tested for red desaturation and describes his deficit as 20% in his right eye. The significance of this test is that decreased red colour perception is a sign that there may be macula or optic nerve abnormalities. Red colour perception is an early sign of potential problems, even when visual acuity appears unaffected. Manuel was tested for relative afferent pupil defect (RAPD), presumably to determine if there were changes in the macula, retina or optic nerve that prevented normal bilateral pupil responses. Manuel's RAPD was deemed to be negative (Chang 2008).

The medical officer recorded Manuel's anterior chambers as deep and quiet. A Goldman tonometer was applied and he had a bilateral intraocular pressure reading of 9 mmHg. So far, results confirm that the problem is likely to be posterior to the lens and that intraocular pressure is not a mitigating cause for his vision loss. Manuel was dilated and ophthalmoscopy performed. A small amount of cells were noted in his right vitreous. Further, his right fundus showed some bleeding around his macula and a pink optic disc. His left fundus showed a small grey scar defined with a black border in his upper left quadrant.

The medical officer provisionally diagnosed 'right active macular toxoplasmosis' and 'possible left ocular toxoplasmosis scar'. The presence of lesions in both eyes and a well observed retinochoroid scar in the left eye, accompanied with blurred vision and floaters in the right eye is consistent with ocular toxoplasmosis (Muccioli et al 2007).

Adding to the strength of this diagnosis is the knowledge that Manuel was born in Venezuela, and *T. gondii* infection rates in South America are estimated between 50 - 80% (Hill et al 2002). *T. gondii* is known to infect water sources in South America (Dodds 2006). Manuel may have contracted *T. gondii* congenitally, or acquired it through drinking water at a later age. Manuel is in the typical age bracket of 25 - 45 years, which is the classical period for reactivation of retinochoroidal scars (Ng & McCluskey 2002).

Diagnostic Methods

The paramount diagnostic method was fundal examination. This enabled lesions and their location to be observed. The medical officer decided to confirm the diagnosis of ocular

toxoplasmosis and rule out syphilis by ordering blood be sent to the laboratory for *T. gondii* IgG and IgM, and *Treponema pallidum* antibody detection. Manuel tested positive for *T. gondii* IgG antibodies, but negative for IgM and *Treponema pallidum* antibodies. The significance of these results are discussed later, under the subheading **Immune Response**.

The medical officer also ordered an immunology test for autoimmune markers. In 1998 Manuel was tested for antinuclear antibodies (ANA), presumably around the time he was diagnosed with optic neuritis. In 1998 he had a negative result. His latest results are now positive for ANA. ANA presence may be an indication that Manuel has an autoimmune disease of connective tissue, such as systemic lupus erythematosus (SLE). However, ANA presence may be positive in healthy people, or induced by transient infection, and does not constitute a diagnosis of SLE in the absence of systemic signs and symptoms (Brighton & Sussex 2008). There is no evidence that Manuel has exhibited any other systemic symptoms indicative of SLE.

Later, as an inpatient, the medical officer decided to conduct a vitreous tap and send the the specimen to the laboratory. *T. gondii* is rarely observed in intraocular fluids and vitreous taps carry risks. It is not normally recommended as a routine diagnostic method (Muccioli et al 2007). For this reason, the vitreous tap was only used when Manuel's symptoms were worsening. The test was trying to isolate herpes simplex virus, herpes zoster virus and cytomegalovirus. These viruses are known to cause diffuse retinochoroiditis but do not normally cause focal lesions (Lightman & Towler 1998: 112). Manuel's vitreous was negative for all three viruses.

Immune Response

Manuel's serology results indicated that he had *T. gondii* IgG antibodies. However, he did not have any IgM, or *Treponema pallidum* antibodies. When *T. gondii* enters the body, there is an innate and generic immune response whereby macrophages attempt to phagocytose *T. gondii*. This process results in the release of interleukin from the macrophages which stimulate the proliferation of T Cells, which in turn stimulates the proliferation of B Cells. B Cells differentiate into plasma cells, which in turn produce antigen-antibody complexes, or immunoglobulins. On primary exposure to *T. gondii*, IgM antibodies rapidly increase in number to combat the infection. IgM antibodies shortly return to negligible levels thereafter. In the mean time, IgG antibodies which are more specific and longer lasting, slowly increase in numbers. IgG antibodies linger and remain in a constant state of readiness for subsequent exposure, or reactivation of *T. gondii*.

Serologic testing has limitations and does not constitute a diagnosis on its own. The presence of *T. gondii* IgG antibodies merely means that at some point, Manuel has been infected by *T. gondii*, either congenitally, or acquired. Owing to the fact that most active

retinal lesions are reactivations of prior disease, often there is an absence of T Gondii IgM antibodies (Muccioli et al 2007).

Immune responses cause degradation of the retina and choroid. Manuel has an active lesion affecting his macula in his right eye. Generic and specific immune responses result in the isolation of active parasites and subsequent transformation into inactive parasitic cysts. Active lesions become less fuzzy and more defined as immune responses subside. The borders of these lesions may become hyper-pigmented (Muccioli et al 2007). Manuel has an inactive lesion in the upper left quadrant of his left retina. Breakdown of the cyst walls transforms inactive parasites into active parasites and reactivates the lesions (Koo et al 2006).

Management, including medical and psycho-social

Predominantly, Manuel's management has been carried out in the outpatient setting. During the course of his illness, he spent a six day period in the inpatient setting. Medically, his management involved a combination of pharmacotherapy, which will be discussed later under **Pharmacology**, and fundal surveillance. Ocular toxoplasmosis is normally self limiting in immunocompetent people and not all episodes necessitate therapeutic intervention. However, pharmacotherapy is a well recognised means of controlling the retinochoroidal inflammation and preventing *T. gondii* infection from becoming significantly worse (Levinson & Rikkers 2004).

There is little or no mention made of psycho-social management. The only documented management related to this, involved the provision of a medical certificate, presumably for Manuel's employer. Manuel lives at home, and I witnessed obvious family support. However, Manuel is a young fit man who woke up one morning, and without notice, had blurry vision. The impact this disease process has had on his life is real and debilitating. Any patient that experiences significant visual loss, will require practical and emotional support to deal with the imminent change to their life, regardless of the cause and timeframe (Stollery et al 2005: 2).

Pharmacology

Manuel was treated with a variety of different drugs during both his time as an outpatient and an inpatient. The potential benefits of therapy need to be weighed against the potential side effects from therapy (Levinson & Rikkers 2004). Predominantly, Manuel was treated with a combination of antibiotics and steroids, both as oral preparations and eye drops. The standard treatment for ocular toxoplasmosis includes systemic antiparasitic drugs used with or without corticosteroids (Koo et al 2006).

It is widely accepted that the antiparasitic drug of choice in treating ocular toxoplasmosis is pyrimethamine. However, Manuel was treated with oral clindamycin. In a systematic

review of the literature in the use of antibiotics in the treatment of ocular toxoplasmosis, there was a lack of evidence to support the use of routine antibiotic therapy. Further, there was weak evidence that the use of long term antibiotics for chronic recurrent ocular toxoplasmosis reduced the recurrence (Gilbert et al 2008). However, according to Australian authors, clindamycin used solely or in conjunction with corticosteroids has had good results (Ng & McCluskey 2002). Pyrimethamine must be taken in conjunction with folic acid supplements and requires careful blood monitoring (Koo et al 2006). Clindimycin does not require stringent monitoring, however it can produce pseudomembranous colitis, intolerable nausea and vomiting (Sobrin et al 2007).

Corticosteroids are used to reduce the damage caused by inflammation. Because corticosteroids can mask the inflammation caused by *T. gondii*, it is recommended that corticosteroids never be used as a single treatment regime, and that treatment with antiparasitic drugs commence prior to steroid treatment (Levinson & Rikkers 2004). Corticosteroids have their own risks and must not be stopped without weaning. There is evidence that Manuel was prescribed a weaning regime. During Manuel's time as an inpatient he was treated with both oral and intensive topical prednisolone.

In the absence of marked improvement in his lesion, Manuel was additionally given intravenous acyclovir, during his inpatient stay, whilst the results of his vitreous tap were pending. Acyclovir is not therapeutic against *T. gondii* as it is an antiviral agent and not antiprotozoal. Acyclovir would be therapeutic if he turned out to have herpes simplex or herpes zoster, but he did not. Treatment with acyclovir ceased as soon as herpes simplex and herpes zoster were ruled out as possible causes (Mims Online 2008).

Many times during his numerous visits to the outpatient clinic, Manuel was given tropicamide to induce mydriasis and cycloplegia to facilitate ophthalmoscopy and fundal examination. Tropicamide is the drug of choice because it has a fast onset and is short acting (Mims Online 2008).

Complications

There is no documented evidence that Manuel suffered any complications from his ocular toxoplasmosis, other than decreased visual acuity. On initial presentation Manuel's visual acuity was RVA 6/9-1 LVA 6/4.5-1. Within three weeks he had rapidly deteriorated to RVA 6/60 LVA 6/6-2. At the time I saw him on his most recent consultation, some six months after initial presentation and diagnosis, he had only recovered to RVA 6/21 LVA 6/4.5+2. Central vision loss may be permanent since his lesion involves the macula (Muccioli et al 2007).

The most obvious complication of ocular toxoplasmosis is lesion recurrence and decreasing loss of vision. Other less common complications include sub-retinal neovascularisation and retinal detachment (Muccioli et al 2007).

Legal and Ethical Aspects

There are many angles from which Manuel's care and management are governed by overriding legal considerations. However, for the purpose of this case study I have focused on one key area that is relevant. Documentation is a fundamental aspect of the ophthalmic nurse's role. Documentation is vitally important as it is a means by which the wider healthcare team communicates. It may also be called upon for research and teaching purposes such as this, or as evidence in a court of law. When documenting, nurses need to be aware of several essential criteria that should be met. Documentation should be accurate, legible, objective and timely. Abbreviations should be limited and medical terminology correctly used. Further, incorrect entries should be legible and entries should only be made by self for self (Staunton & Chiarella 1997: 171-175). There is good evidence in Manuel's progress notes that the nursing staff have been documenting appropriately. As I researched this paper, I did not encounter any difficulty following Manuel's progression from initial presentation to his most recent clinic consultation.

There are also many angles from which Manuel's care and management are governed by overriding ethical considerations. For the purpose of this case study I have focused on four common bioethical principles. The first principle is autonomy. This principle recognises that Manuel possesses the right to control when and what happens to him. Manuel presented to the emergency department of his own volition. There is a presumption that he as a self determining being, is not being coerced into any investigative or treatment modality. One example of this is the written evidence that outlines the discussion, and his subsequent agreement to have a vitreous tap performed upon him. The second principle is non malificence. This principle dictates that no harm should be done to Manuel. I saw no evidence that harm was done to Manuel during the progress of his condition. Thirdly, is the principle of beneficence. This principle dictates that good should be done to Manuel. There is evidence that good was done to Manuel during the progress of his condition. Interventions such as corticosteroid pharmacotherapy, undeniably had a beneficial affect on Manuel. Whilst antibiotic pharmacotherapy may or may not have done Manuel good, it certainly did him no harm. Lastly, is the principle of justice. This principle dictates that Manuel should be treated fairly, and that every opportunity should be afforded to him in an equivalent way to anybody else (Staunton & Chiarella 1997: 28-30). I saw no evidence that Manuel was discriminated against.

Nursing Care Plan

Ophthalmic nurses need to be able to assess and plan patient care on an individual basis (Stollery et al 1998: 16). The nursing staff that looked after Manuel in the inpatient setting have initiated daily blood sugar level readings. Manuel received high doses of both oral and topical prednisolone. Prednisolone is a synthetic glucocorticoid and was used to treat the inflammation of his retinochoroiditis. Glucocorticoids have serious systemic effects including hypertension, diabetes and acute psychosis (Lightman & Towler 1998: 123).

Manuel's blood sugar levels averaged 6.1 mmol/L, and his systolic blood pressure never rose above 130mm/Hg during his inpatient stay. There was no reported evidence of a psychotic episode.

Outcome

The clinical outcome for Manuel is that he is still suffering from decreased visual acuity in his right eye, currently 6/21. Recent fundal photographs in the outpatient department have shown that the lesion affecting his right macular has now scarred. It is unlikely that Manuel will recover 6/6 vision in his right eye (Levinson & Rikkers 2004). Treatment may have helped to control Manuel's clinical episode, but it in no way prevents him from getting likely recurrences (Ng & McCluskey 2002). Manuel is back at work, and is still under the care of the retinal clinic at Sydney Eye Hospital. He is no longer taking pharmacotherapy. At his last clinic consultation, he was advised to return to the emergency department if he noticed any further deterioration in his vision. Manuel is due to return to the retinal clinic in November 2008. If there is no further vision deterioration than he will be discharged from the care of the clinic. I shall endeavour to be present at Manuel's next clinic consultation, or at least follow up on his medical record to ascertain to what extent this disease process is still affecting him.

Conclusion

At the beginning of this case study I outlined what I hoped to achieve in researching its contents. Firstly, I hoped to gain an understanding of toxoplasmosis, and in particular ocular toxoplasmosis. In short, toxoplasmosis or ocular toxoplasmosis is the name given to the clinical manifestations of people who are infected by the protozoa *Toxoplasma gondii*, either systemically or ocularly. Secondly, I aimed to gain an understanding of Manuel's story. It is probable that through no fault of his own, Manuel is likely to suffer with ocular toxoplasmosis for the rest of his life. The disease has had real and devastating effects on his vision, and those are likely never to fully improve. In the end, it is left up to Manuel to try and adapt to his new vision as much as possible with the aid of his family, friends and the wider healthcare team at Sydney Eye Hospital.

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