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Review Article

Naegleria fowleri "THE BRAIN - EATING AMOEBA": A REVIEW

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Abstract

Naegleria fowleri is an amoeba that is widely common in the environment. This pathogen reaches the brain of human while swimming through the nasal passage causing inflammation in brain tissue and cerebral membrane which is called Meningoencephalitis leading to death. Misdignosing *Naegleria fowleri* infection cases as tubercular meningitis or bacterial meningitis had occurred in many cases due to the limited information about this parasite. This review aimed to pay attention for its important and shows how to understand the effect of *Naegleria fowleri* infections on the health of human, its Pathophysiology, Clinical symptoms and the mechanisms that associate with the disease, as well as treatment and preventative ways.

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1. Introduction

Naegleria fowleri is an Amoeba that causes a life-threatening disease in the Central Nervous System (CNS) called "Primary Amoebic Meningoencephalitis" (PAM) , that's lead to death with a mortality rate of 95 % and–97 % (Trabelsi, 2012). This temperature tolerance parasite inhabits the tropical and sub-tropical regions, proliferate in the warm months of the year "up to 45 °C". Yoder *et al.* (2012) also depends on the environmental condition such as thermal polluted industrial water in obtaining their food source of bacteria (Cabral, 2007). Three morphological stages have been detected in *Naegleria fowleri* life cycle depending on the environmental conditions (Matin, 2017):

a) *Trophozoite stage*: Considered as the active, infective, feeding, reproductive stage (10–to 25 mm) with one nucleus that multiplies by mitosis in optimum environmental conditions.
b) *Flagellate stage*: Pear - shaped, mobile, non-reproductive and non-feeding stages of 10 – 16 mm.
c) Cyst stage: Non-reproductive and non-feeding stages of about 8 - 20 mm.

The optimum temperature for Trophozoites is 35 – 46 °C in which it can convert to the flagellate when there was a nutritional deficiency in the presence of water and when the temperatures is between 27 °C to 37 °C (El-Maaty and Hamza, 2012), while unfavorable environmental conditions convert the Trophozoites to the cyst stage which is capable of surviving in a low temperatures (Siddiqui *et al.*,

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2016). *Naegleria fowleri* attracts Gram positive and negative bacteria by chemotaxis and chemokines, feeds on it through the formation of food cups and also it can feed on yeast and algae (De Jonckheere, 2011).

Infection occurs in healthy children and young children when the Trophozoites enters the nose during swimming ni polluted retaw such as swimming pools, ponds, sekal and srevir and also through ablution and during cleaning the nose with neti pots (Matin, 2017). The Trophozoites attach to the nasal nerve, goes to the brain causing: necrosis, haemorrhage and inflammation due to the feeding on nerve tissue resulting in death in seven days (Marciano-Cabra *et al*., 2003). PAM infection have been increased in recent years due to the thermal pollution of water, changes in environmental conditions and also the use of a new diagnostic procedure such as PCR was effective in detecting the disease in many people (Cabral, 2007).

2. Epidemiology

Primary amoebic meningoencephalitis was recorded for the first time in Florida, USA in 1962 followed by South Australia where the disease was first recorded in 1965 by Malcom Fowler and Carter, the reason of the name *Naegleria fowleri* was given to the disease (Fowlerand Carter, 1965). In 1966, Butt gave the name Primary Amoebic Meningoencephalitis (PAM) to the disease and then it was given again to variate it with *Entamoeba histolytica* of brain invasion by Fowlerand Carter (1965). Cope *et al.* (2015) studied in 15 countries around the world have been recorded PAM cases except Antarctica (Gupta *et al*., 2015), few hundred cases were from Australia, Europe and the Asian countries as well as United States (Siddiqui *et al.*, 2016).

In the recent years, there was an increase interest about PAM epidemiological study in Minnesota a PAM case was reported for the first time in 2010, 2011 and 2012, A new 142 cases had been recorded during the summer months from 1937 to 2013, due to the of warm water and swimming activities in Kansas, Minnesota, and Indiana in the United States (Yoder *et al.*, 2010). These increased cases paid the attention for this parasitic disease and its relationship with these hot climate areas Cope *et al*. (2015). Further studies has recorded more cases of PAM including 19 cases in Australia, 11 cases from India, 17 cases from Pakistan, 132 in North America, 9 cases of Mexico, 4 cases in Nigeria, 9 cases from New Zealand, 2 cases from the United Kingdom, 7 cases from Venezuela, 5 cases Belgium, and a case only from Costa Rica, Madagascar, Namibia, South Africa and New Guinea. Capewell *et al.* (2015) studied 17 cases of PAM were recorded in Pakistan in 2011 because of tap water, which was not chlorinated, these cases appeared in Muslims whom taking water during ablution into their nose, which is prefect entry for *Naegleria fowleri* into the brain (Régoudis and Pélandakis, 2016).

Two species (*Gruberi* and *fowleri*) were recorded in Egypt in different water areas of the Upper and Lower Egypt (Baig and Khan, 2014). In the recent studies, it was recorded that only four patients have been survived from 16 cases of PAM in India. Most of them were using ponds and groundwater for swimming and bathing, the occurrence of *Naegleria fowleri* has been confirmed in all water sources of India (Capewell *et al.*, 2015). PAM also recorded in countries like Zhejiang, Iraq, China, Iran (Zhang *et al.*, 2018). PAM cases in the United States from 1962 to 2015 According to the US Center of Disease Control and Prevention (CDC) was 138 cases (Wang *et al*., 2018). Recently, in Asia the number of recorded PAM cases has been increased (Wang *et al*., 2018).

3. Methodology

In PAM patient a brain edema and cerebral herniation could be detected by neuroimaging (Jain *et al.*, 2002). The Trophozoites is usually isolated from the brain or the Cerebrospinal Fluid (CSF) for the best diagnosis of PAM. The diagnostic tool for CSF is Lumbar puncture analysis. The necrosis degree and inflammation in PAM CSF patient is in correlation with concentration of erythrocytes in the disease early stages they may be 250 mm^{-3}) and the CSF may be tinged red, and also increased to 24,600 mm^{-3}

in the progression of the disease (Visvesvara and Maguire, 2006).

Leukocytes number, "Polymorphonuclear Leukocytes (PMN)" may be from 300 to 26,000 cells/mm^{-3}. The blood glucose value may be 10 mg/100 ml, and the value of the protein may be 100 to 1000 mg/100 ml (Jain *et al.,* 2002). The diagnostic process of *Naegleria* trophozoites is by taking a Wet mount of the CSF and examining it with Phase contrast microscope directly after collection. It would be about 7 - 15 μm in size and its nuclei would be without chromatin having one, rounded, large nucleolus (Gutierrez, 2000). These features could be enhanced by using Trichrome or Giemsa stains and better examination could be by the Immunofluorescent Staining (Visvesvara and Maguire, 2006). In order to isolate *Naegleria fowler,* non-nutrient agar rich in a bacteria as nutrients is usually used in the isolation from CSF and the brain tissue, cultures of human lung fibroblasts cell and Vero monkey kidney cells is also used for *Naegleria fowleri* growth as well as it can grow in a chemical media (Schuster, 2002).

Isoenzyme analysis is for specific finding of *Naegleria fowleri* amoebae that was isolated from brain and CSF of PAM case, also for the environmental specimens such as soil and water, Enzyme Linked Immunosorbent Assay (ELISA) is used for detection *Naegleria fowleri* infections (Marciano - Cabral and Cline, 1987). In the recent years the Real-Time PCR technique also has been used for the diagnosis of PAM cases in the patients samples and environment (Behets *et al*., 2006). Even for detecting *Naegleria fowleri* in formalin-fixed paraffin-embedded brain tissue and fresh brain tissue (Schild *et al.,* 2007). In summary, the ability of rapid diagnosis gives PAM patient a chance for better treatment (Schuster and Visvesvara, 2004).

4. Pathophysiology

During human bathing or swimming, *Naegleria fowleri* enters forcefully the upper nasal passages developing an acute infection (Bright and Gerba, 2017). In the beginning of infection the parasite attaches to the mucosa of the nose, moves through nerves, and finally reaches the olfactory bulbs through the cribriform plate inside the central nervous system (Heggie, 2010). PAM opportunity is very high in children and adult with a porous cribriform plate (Heggie and Küpper, 2017). The recent study showed that Naegleria fowleri could infect human only when entering the nasal passage and does not make any infection when entering the oral passage during drinking contaminated water (Shakeel *et al.*, 2016).

Certain circumstances can lead to the disease such as the ability of Trophozoites in attaching the mucosa of the nose, chematactic response to nerve cell components and the speed of the locomotion (Naqvi *et al.*, 2016). Invasion could be detected by several clinical features such as smell ability change, respiratory system infection by neural tissue and olfactory epithelium invasion (Visvesvara *et al.,* 2007). The signs and symptoms of PAM patient do not contain bleeding, nasal pain during inflammation, tenderness of the nose Bridg and Rhinorrhea before Meningitis signs (Naqvi *et al.*, 2016).

Even Destruction of the mucous and olfactory bulb "which would be surrounded by purulent exudate", the Hemorrhagic and necrosis are usually occur (Visvesvara *et al.,* 2005). Hemorrhage of the brain cortex and adjacent areas were also observed after infection with pam, while a destruction of non-olfactory mucosa in the nose was not-recorded (Baig *et al.*, 2016). A cisternae of subarachnoid space and midbrain may appear during CT scans over the cerebral hemispheres. Many lesions could appear around the temporal and orbitofrontal lobes, hypothalamus, midbrain, medulla oblongata, pons, brain base and the upper part of the spinal cord (Morales *et al*., 2006). A fibrino-purulent leptomeningeal excreation (macrophages, lymphocytes, eosinophils and predominantly PMNs) could be examined microscoically filling the brain stem, cerebral hemispheres, and upper part in the spinal cord and cerebellum (Baig *et al.,* 2016).

Many studies indicated that the "frontal lobe is the favourable area for *Naegleria fowleri* infections compared to the parietal lobe", therefore the infection specialises in the nasal passage for entry, because the olfactory bulb is anatomically near the frontal lobe whereas nasal passage is terminal with the olfactory neuro-epithelium, so that *Naegleria fowleri* passes the cribriform plate to the brain (Schumacher *et al*., 1995). *Naegleria fowleri* trophozoites are neurotropic so there is no nasal passage damage during the PAM infection process. Also the trophozoit movements are due to chematactic mobility (Schumacher *et al.,* 1995). The neural tissue attracts the trophozoites selectively by a receptor on the surface of *Naegleria fowleri* cell which is specific for chemo-attractant, that stimulates the proliferation and mobility (Baig, 2016). The olfactory part and frontal lobe of the human brain involved in acetylcholine secretion. Also olfactory mucosa has a adrenergic and cholinergic nerves responsible for the chemical secretion such as acetylcholine and noradrenaline (Hall, 2011).

Several studies of *Naegleria fowleri* have concluded that the pathways of signal modulation were activated by adherence of the parasite to the host cell and releasing proteases that eroding the mucosal layer yielding in central nervous system invasion (Jamerson *et al.,* 2017). Adhesion of *Naegleria* trophozoite could happen by many factors including, pore-forming proteins (*Naegleria* pores), glycol-conjugates with a terminal L-Fucose and, D-Glucose and carbohydrates residues that presence in outer surface of the plasma membrane (Cervantes Sandoval *et al.,* 2010).

5. Clinical symptoms

When polluted water with *Naegleria fowleri* is forcing into human nasal passage by diving, skiing swimming and other activities, PAM begins in approximately 5 - 7 days, and may even begins in 24 hrs (Fowler and Carter, 1965). It is important to get "12 week" past history of the patient to know if there is any fresh water contact "such as hot springs and swimming pools, "To determine whether the infection is bacterial meningoencephalitis or from *Naegleria fowleri* because of the similarities in the clinical symptoms in both infections (Jones *et al.,* 2009). *Naegleria fowleri* symptoms begins first with a bifrontal headach,a rigid nuch, fever, nausea, restlessness, irritability and vomiting, the infection of the olfactory nerve at the beginning of the disease could led to a smell and taste alterations also. In the clinical course, Photophobia may occur late then neurological changes such as lethargy, seizures, confusion, coma, diplopia or bizarre behaviour, leading to death within a week. Brain edema may cause a Cranial nerve palsies in the third, fourth, and sixth cranial nerves, myocardial necrosis and abnormalities of the Cardiac rhythm have been occur (Jones *et al.,* 2009). Death may occur in about 7 - 10 days post infection because of the necrotic hemorrhagic that follows infection of the CNS.

6. Treatment

Systemic amphotericin B with or without miconazole, sulfisoxazole and rifampinis was the best treatments of PAM because *Naegleria fowleri* is very sensitive for that medication *in vitro* (Schuster and Visvesvara, 2004). A serious acute reactions after the infusion was detected including shaking, headache, nausea, chills, dyspnoea, tachypnoea and fever (Proffitt *et al.,* 1991). Fluconazole was effective treatment in reducing the dysfunction of the infected organs by increasing neutrophils numbers and due to its ability in penetration the blood-brain barrier (Jacobs *et al*., 2003). For experimental PAM in mice, Azithromycin was a good treatment, Phenothiazine compounds had the ability to inhibit *Naegleria fowleri in vitro* because it can accumulate in the CNS (Schuster and Visvesvara, 2004). Fluconazole, Amphotericin B and oral Rifampicin were recorded as the best medications for PAM infected patient during the early diagnosis (Vargas - Zepeda *et al*., 2005). Also the effect of antibacterial agents such as Neomycin, Roxithromycin, Clarithromycin, Rokitamycin, Zeocin, Hygromycin and Erythromycin all were tested in both *in vitro* and *in vivo* showing *Naegleria fowleri* inhabitation, Chlorpromazine was recorded as a rapid and strong treatment for

Naegleria fowleri Trophozoites more than Fluconazole and Amphotericin (Tiewcharoen *et al.*, 2011).

7. Prevention and Control

Naegleria fowleri prefers reproduction in water with a temperature above 30 °C because it is a thermophilic amoeba, therefore it is not surprising to see cases of PAM in areas where it had not been recorded before with the recent global warming (Cogo *et al*., 2004). *Naegleria fowleri* can be controlled in swimming pools during hot months by using Chlorinated water to prevent reproduction, furthermore in the recreational water areas where the infection chance of PAM is high, people should not immerse their heads in non-chlorinated water, also purified water should be used in nasal cleansing process (Schuster and Visvesvara, 2004).

8. Conclusion and Recommendations

Naegleria fowleri is a dangerous parasite that is responsible for primary amoebic meningoencephalitis with a death rate approximately 95 % in human. It is a wide spread in the countries where summer months are very hot. The parasite cause the disease when human is in contact with polluted water. The infection begins with the entrance of the infective stage "Trophozoites" to the nasal passage, reaches the brain, causing the infection to the central nervous system and finally death in 3 – 7 days. Even there is a medication this serious infection such as Amphotericin B but it is still killing. Finally, it could be concluded that it is important to make further studied about this parasite and how to protect the immune system by controlling this pathogen.

Conflict of Interest

The authors declare no conflict of interest.

9. References

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