Balamuthia mandrillaris

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Balamuthia mandrillaris is a free-living amoeba that is known to cause the deadly neurological condition known as granulomatous amoebic encephalitis (GAE).[1] *B. mandrillaris* is found in the soil and was first discovered in 1986 in the brain of a baboon that died in the San Diego Wild Animal Park. *B. mandrillaris* can infect the body through skin wounds or by inhaling the dust containing *Balamuthia*.[2] *Balamuthia* has not been definitively isolated in nature, but it is believed to be distributed throughout the temperate regions of the world. This is supported somewhat by the presence of antibodies to *Balamuthia* present in healthy individuals. The *Balamuthia* genus is named in honor of the late parasitologist William Balamuth (1914–1981) for his contributions to the studies of parasitic and free-living amoebas.

Balamuthia mandrillaris	
Scientific classification	
Domain:	Eukaryota
(unranked):	Amoebozoa
(unranked):	Discosea
Order:	Centramoebida
Family:	Balamuthiidae
Genus:	***Balamuthia*** Visvesvara et al., 1993
Species:	***B. mandrillaris***
Binomial name	
Balamuthia mandrillaris Visvesvara et al., 1993	

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Morphology

Balamuthia mandrillaris is a free-living, heterotrophic amoeba, consisting of a standard complement of organelles surrounded by a three-layered cell wall, and with an abnormally large cell nucleus. On average, a *Balamuthia* trophozoite is approximately 30 to 120 micrometres in diameter. The cysts fall approximately in this range as well.[3]

Life cycle

Balamuthia's life cycle, like the *Acanthamoeba*, consists of a cystic stage and a trophozoite stage, both of which are infectious, and both of which can be identified as inclusions in the brain tissue on microscopic examination of brain biopsies performed on infected individuals. The trophozoite is pleomorphic and uninucleated, but binucleate forms are occasionally seen. Cysts are also uninucleated possessing three walls: an outer thin irregular ectocyst, an inner thick endocyst, and a middle amorphous fibrillar mesocyst.[4]

Pathology

Balamuthia mandrillaris is larger than human leukocytes therefore making phagocytosis impossible. Instead, the immune system attempts to contain them at the portal of entry by mounting a type IV hypersensitivity reaction.[5] They may enter the body through the lower respiratory tract or through open wounds. Upon introduction, the amoeba may form a skin lesion, or may migrate to the brain, causing a condition known as granulomatous amoebic encephalitis,[6] (GAE), which is usually fatal. This granulomatous feature is mostly seen in immunocompetent patients; immunocompromised individuals exhibit a "peri-vascular cuffing".[7] *Balamuthia*-induced GAE can cause focal paralysis, seizures, and brainstem symptoms such as facial paralysis, difficulty swallowing, and double vision.

Balamuthia may also cause a variety of non-neurological symptoms, including skin lesions, which can progress to GAE. Patients experiencing this particular syndrome may report a skin lesion (often similar to those caused by MRSA), which does not respond well to topical antibiotics. The lesion is usually localised and very slow to heal, or fails to heal altogether. In some presentations, the infection may be mistaken for certain forms of skin cancer or leishmaniasis. *Balamuthia* lesions on the face often cause swelling.

Culturing and identification

Balamuthia is most easily identifiable in a brain biopsy performed on an individual suffering from GAE. The amoeba cannot be cultured on an agar plate coated with gram negative bacteria because unlike Naegleria, *Balamuthia mandrillaris* does not feed on bacteria. Instead *Balamuthia* must be cultured on primate liver or human brain microvascular endothelial cells (the cells that constitute the blood–brain barrier).[8]

Treatment

Balamuthia infection has had successful treatments. In two cases, both were treated with a cocktail of antibiotics and antifungal drugs, although it is unclear if any or all of these medications played a part in treatment. Both victims suffered permanent neurological deficits as a result of their infection. Another two cases were presented and both of these individuals received successful treatments due to discovering the infection early. Two individuals, a five-year-old girl and a 64-year-old man, developed GAE. After diagnosis, they were given powerful antimicrobial therapy. Both patients recovered.[9]

Organ transplantation

According to a MMWR report published in September 2010, two confirmed cases of *Balamuthia* transmission occurred through organ transplantation in December 2009 in Mississippi. Two kidney recipients, a 31-year-old woman and a 27-year-old man, suffered from post transplant encephalitis due to balamuthia. The woman died in February 2010 and the man survived with partial paralysis of right arm. The CDC was notified by a physician on December 14, 2009 about possible transplant transmission in these two patients. Histopathologic testing of donor and recipient tissues confirmed the transmission. Two other patients who received heart and liver transplants from the same donor but in different hospitals were placed on preemptive therapy and remain unaffected. A second cluster of transplant transmitted *Balamuthia* in Arizona was reported in the same weekly report. There were four recipients: two from Arizona (liver and kidney-pancreas), one from California (kidney) and another from Utah (heart). Recipients from Arizona—a 56-year-old male and a 24-year-old male—both succumbed to encephalitis within a span of 40 days from transplantation. The other two were placed on preemptive therapy after the first two were reported and remain unaffected.

References

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External links

- http://www.cdc.gov/balamuthia/index.html for images: Cyst of *B. mandrillaris* and Trophozoite of *B. mandrillaris* in culture. Credit: DPDx