Creutzfeldt - jakob disease research paper sample

Environment, Animals

Creutzfeldt - Jakob disease

Creutzfeldt - Jakob disease (CJD) is one of the rarest and fatal neurological conditions that affect both man and animals. It is the most common of human prion diseases. This viral disease as caused by components that are yet to be fully identified. It is majorly believed to be caused by an infectious prion protein. Prion proteins are found in the cells of the human body in normal conditions. In this normal form, prion protein has no harm to human health. However, during coding mutation, prion protein undergoes some changes and become infectious. This infectious form of prion protein is believed to be the main cause of CJD and other related variant diseases. This infectious form of prion proteins causes abnormal accumulation of prion proteins in the nervous system beyond the capability of the body defense system to break them down. The disease is highly contagious and incurable (Cyngiser, 2008, p. 198).

The incurability of the disease springs from the fact that prion proteins do not have nucleic acids, that is, RNA and DNA. This makes CJD defiant of traditional methods of identification and destruction of viruses. Other diseases caused by infectious prion proteins include Fatal Familial Insomnia (FFI), Kuru and Gerstmann-Straussler-Scheinker Syndrome (GSS). FFI and GSS are hereditary disorders but are also to occur spontaneously in rare occasions. Kuru is unique to Papua New Guinean people resulting from their ancient practice of cannibalism and exposure to the infected brain of their human preys. Bovine Spongiform Encephalopathy (BSE) is a form of neurodegenerative disease similar to Creutzfeldt-Jakob (CJD) common in

animals. It is commonly referred to s mad cow disease (Cyngiser, 2008, p. 200).

Types of Creutzfeldt - Jakob disease (CJD)

There are many types of CJD. The most common one is the sporadic type. Others include familial and acquired CJDs. The sporadic CJD is the most popular of the cases, which occur without any clear predisposing conditions to date. It accounts for up to 85% of all CJD diagnosis. It is still a rare case with the prevalence of about two cases in a population of one million per annum. The population at risk is those aged between 55 to70 years. Up to 90% of patients infected with sporadic CJD die within their first year of infection. The clinical and diagnostic symptoms of sporadic CJD are rapid and progressive decline in cognitive abilities of the patient and pseudoperiodic Synchronous discharges which manifests itself through myoclonus. It also includes ataxia on electroencephalogram, and positive results of CSF14-3-3 protein test (Takashi et al, 2009).

Familial CJD is the second most popular of CJDs with prevalence of about 15% of CJD diagnosis. It is hereditary in causation with its patients testing positive for coding mutation on chromosome 20. Chromosome 20 is the genetic character responsible for the control of the formation of prion proteins. It has a long incubation period and its victims live with it longer than sporadic case (Cyngiser, 2008, p. 203).

Acquired CJD is the rarest form of CJD with prevalence of less than 1% of CJD diagnosis. It is acquired through various methods such as variant route, which is the transmission through ingestion of BSE, infected animal products, exposure to infected brain through a medical operation procedure and

injections from adulterated hormones from cadavers. Variant CJD is common to people aged below 40 years and is characterized by a long incubation period of up to 10 years. Infectious prion protein materials from people infected with CJDs are found in spleen, lymph nodes and tonsils (Cyngiser, 2008, p. 203).

Symptoms of CJD

Prevention and Treatment

Just like many other viral diseases, there is no research-found treatment for CJD. Measures undertaken in the absence of full treatment involve prevention of further infection and making of infected patients to feel as comfortable as possible. Preventive measures employed involve ensuring that medical personnel carrying out pre-mortem and post-mortem brain biopsy and autopsy are protected from contracting infection, avoidance of animal products from animals with symptoms of mad cow disease, transfusion of blood, which is free from infectious prion proteins, and seclusion of patients diagnosed of CJD. Currently, the patients diagnosed of CJD are administered with opium to relieve pain and create an illusion of comfort.

Other treatments include sodium valproate and clonazepam, which are administered to contain myoclonus. There are other medical tests being studied to enhance early detection of CJD in order to reduce the risks of transfusion of infected blood, and to tame the virus at an early stage. Such tests include diagnostic blood screening for the increase of s100 protein type. S100 protein is found in the brain glial cells. It can increase because of

uncontrolled increase of infectious prion protein. There is also an ongoing research of the ultimate methods of killing neurons in CJD. (Ryan et al, 2011)

Conclusion

Bibliography

Cyngiser, T. A. (2008). Creutzfeltd-Jakob Disease. American Journal of Electroneurodiagnostic Technology , vol 48: 198-208

Ryan et al . (2011). Notification And Support for People Exposed to the Risk of Creutzfeldt-Jakob Disease (CJD) (Or Other Prion Diseases) Through Medical Treatment (Iatrogenically) (Review). London: JohnWiley & Sons, Ltd

Takashi Haraguchi et al,. (2009). Coexistence of Creutzfeldt-Jakob Disease, Lewy Body Disease, and Alzheimer’s Disease Pathology: An Autopsy Case Showing Typical Clinical Features. Neuropathology, vol. 29, 454–459