

Dementia with Lewy Bodies Syndrome

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Received November 19, 2018; Accepted December 12, 2018; Published February 13, 2019

ABSTRACT

Slimming body syndrome with intellectual decline is a genetic disorder of the nervous system that is associated with decreased intellectual function, a group of known Parkinson's motor problems, visual hallucinations, and sudden changes in behavior and sleep disorders. Slimming body syndrome is caused by a mental decline due to the mutation of SNCA genes that is positioned in the long arm of chromosome 4 as 4q22.1 and the SNCB gene, which is based on the long arm of chromosome number 5, is 5q35.2. In addition, GBA gene mutations that are based on the long arm of chromosome 1, 1q22, increase the risk of developing the syndrome, but its main cause is still unclear.

Keywords: Slimming body syndrome, SNCA, SNCB, GBA Genes, Nervous disorder

GENERALIZATIONS OF SLIMMING BODY SYNDROME WITH INFERIORITY

Slimming body syndrome with intellectual decline is a genetic disorder of the nervous system that is associated with decreased intellectual function, a group of known Parkinson's motor problems, visual hallucinations, sudden changes in behavior and sleep disorders. This disorder is commonly affected in older adults, who are often between the ages of 50 and 85. The life expectancy of people with slimming body syndrome varies with mental decline, and people usually die about 5 to 7 years after diagnosing the syndrome [1] (**Figure 1**).

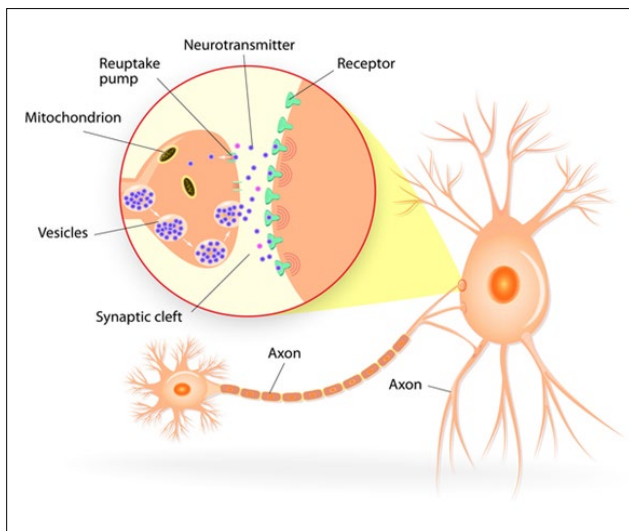


Figure 1. Schematic of the structure of the neuron (neuron).

CLINICAL SIGNS AND SYMPTOMS OF SLIMMING BODY SYNDROME WITH INFERIORITY

Sleep disturbances are usually the first symptom of a slimming body syndrome with rational deterioration. This disorder can occur many years before other symptoms occur. People with this syndrome talk and talk with sleep disorders during sleep, which is a dream narrative of their dream [2].

Racial deterioration is the second most important feature of this syndrome. This reduction in thinking often leads to impairment in geometric and visual work such as puzzle solving. Affected people may also be in trouble in developing problem-solving skills, speech and speech. In people with slimming body syndrome with intellectual decline, visual illusions are created that includes both humans and animals. Behavioral and intellectual fluctuations include a sudden change in attention, thinking processes, and mood [3].

Parkinsonism is usually the last important feature of the body's slimming syndrome with rational deterioration. In people with this syndrome, motor problems usually include vibration, abnormal slow motion (Brady Keynesian) and

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Citation: Asadi S, Amjadi H, Valizadeh G & Jamali M. (2019) Dementia with Lewy Bodies Syndrome. J Ageing Restor Med, 2(1): 64-67.

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and muscle aberration and disturbance (ataxia). It should be noted that damaged people often need wheelchairs to move [3].

Individuals with dementia with dementia may also experience a marked reduction in blood pressure during standing (orthostatic hypertension), syncope, urinary flow control, or constipation [3] (**Figure 2**).

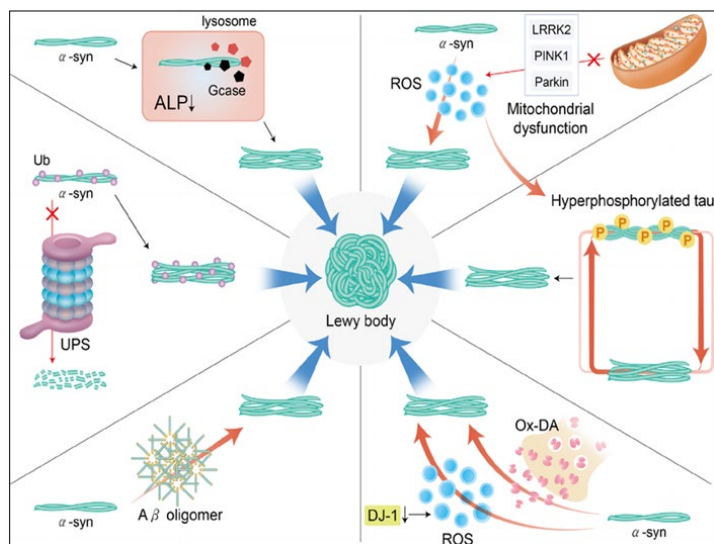


Figure 2. A schematic of signs and symptoms of slimming body syndrome with rational deterioration.

ETIOLOGY OF SLIMMING BODY SYNDROME WITH INFERIORITY

Slimming body syndrome is caused by a mental decline due to the mutation of SNCA genes that is positioned in the long arm of chromosome 4 as 4q22.1 and the SNCB gene, which

is based on the long arm of chromosome number 5, is 5q35.2. In addition, GBA gene mutations that are based on the long arm of chromosome 1, 1q22, increase the risk of developing the syndrome, but its main cause is still unclear [4] (**Figures 3 and 4**).

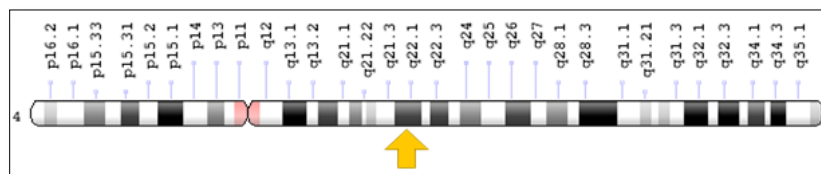


Figure 3. Schematic view of chromosome 4, which SNCA gene is based on the long arm of this chromosome, as 4q22.1.

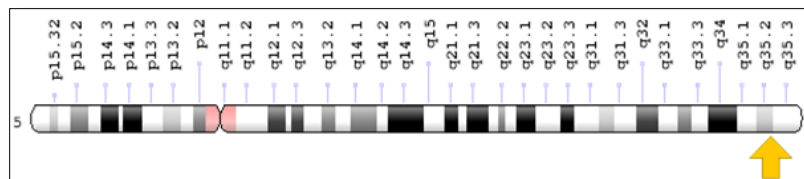


Figure 4. Schematic view of chromosome number 5, where the SNCB gene is located in the long arm of this chromosome as 5q35.2.

SNCA and SNCB genes provide instructions for the synthesis of proteins found mainly in the brain and are also known as alpha-synoclinic and beta-synoclinic, respectively. The alpha-synoclinic protein plays an important role in the communication between neurons (neurons), which helps regulate the release of biochemical messages (neurotransmitters). The beta-synoclinic protein is most

likely involved in a process that allows the neurons to be modified over time, which is essential for learning and memory. Protein beta-synoclinic may also prevent the accumulation of alpha-synoclinic protein in neurons. The enzyme produced by the GBA gene is found throughout the body in cellular structures called lysosomes, which digest and recover proteins and other ingredients [5] (**Figure 5**).

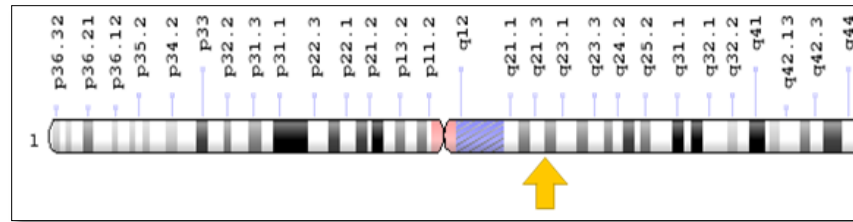


Figure 5. Schematic view of Chromosome # 1 in which the GBA gene is located in the long arm of this chromosome as 1q22.

The mutation in these three genes leads to a slimming body with rational deterioration. SNCA gene mutations disrupt the production of alpha-synoclinic protein, and SNCB gene mutations result in the production of beta-synoclinic modified proteins, which prevents the accumulation of alpha-synoclinic proteins in neurons. GBA gene mutations are thought to disrupt the normal function of lysosomes. Studies have shown that inappropriate lysosomes interfere with the alpha-synoclinic protein degradation and increase the risk of accumulation and slimming [6].

In the body's sluggish syndrome, with intellectual decline, the mutated genes in the brain cause impaired functioning of the neurons and ultimately lead to cell death. Neurons that transmit neuronal dopamine appear to be more susceptible to

sluggish body syndrome with intellectual decline. Dopamine has many important neurological functions, including the role of complex roles in thinking, motivation and behaviour and movement control. Over time, the loss of dopamine-producing neurons increasingly interferes with intellectual and motor function and emotion regulation and creates signs and symptoms of slimming body syndrome with rational deterioration [6].

The body's slimming syndrome follows a morbid decline of the dominant autosomal inheritance. Therefore, to produce this syndrome, SNCA, SNCB, GBA (parent or parent) gene mutations are required and the chance of having a child with this syndrome in the dominant autosomal state is 50% for each possible pregnancy [6] (**Figure 6**).

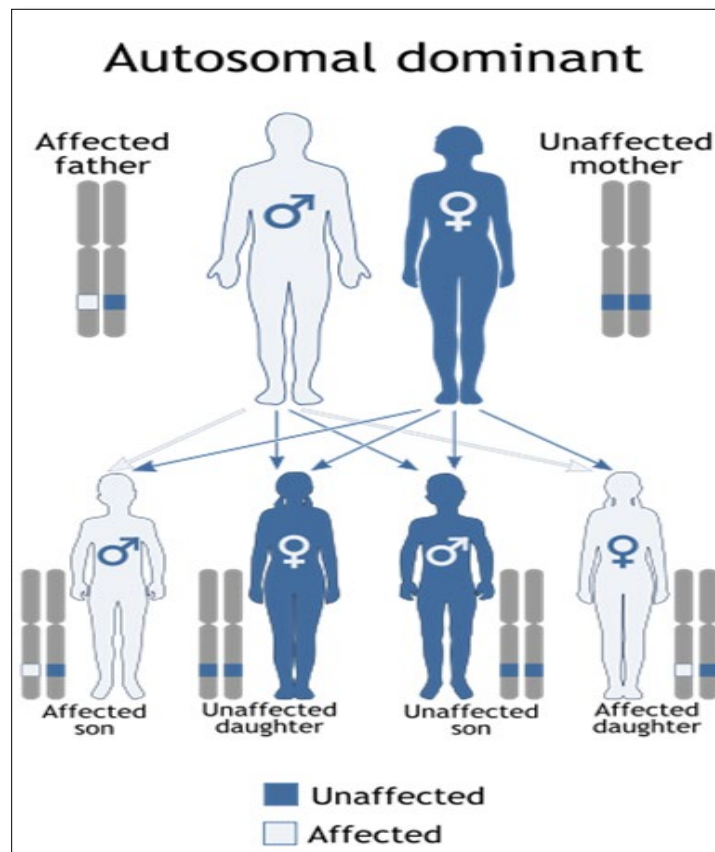


Figure 6. Schematic view of the dominant autosomal inheritance pattern that complicates the body's slimming syndrome with rational deterioration.

FREQUENCY OF BODY WEIGHT LOSS SYNDROME WITH RATIONAL DETERIORATION

The body's slimming syndrome is associated with a mental decline in neurogenic disorder that is estimated to affect about 1.4 million people in the United States. This syndrome accounts for about 5% of mental dementia in the elderly and is the second most common premature neurological disorder after Alzheimer's [6].

DIAGNOSIS OF SLIMMING BODY SYNDROME WITH RATIONAL DETERIORATION

Slimming body syndrome is diagnosed with intellectual decline based on the clinical and clinical findings of the

patients and some pathological and neurological examinations. Electroencephalogram technique is effective in evaluating brain disorders in these patients. The most accurate diagnostic method for this syndrome is the molecular genetic testing of SNCA, SNCB and GBA genes to investigate the presence of possible mutations [6] (Figure 7).

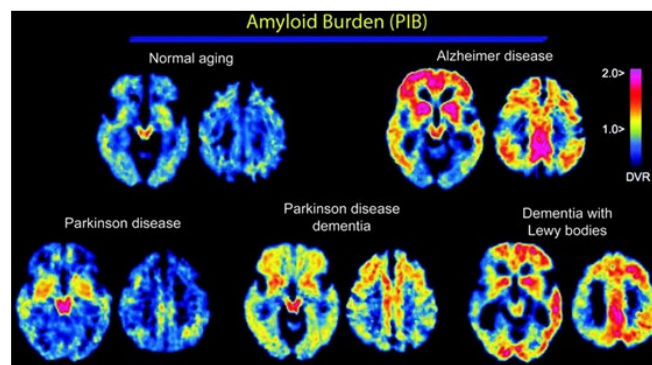


Figure 7. Radiological image of the brain with a slimming body syndrome with intellectual decline compared with other brain disorders.

THERAPEUTIC TRAITS OF THE BODY'S SLIMMING SYNDROME WITH RATIONAL DETERIORATION

The strategy of treatment and management of the body's slimming syndrome is symptomatic and supportive with intellectual decline. Treatment may be done by a team of experts, including a neurologist, psychiatrist, orthopedic specialist, hematologist and other health care professionals. There is no definitive treatment for this syndrome and all clinical measures are needed to reduce the suffering of the sufferers. Genetic counseling is also important for all parents who want a healthy baby [6].

DISCUSSION AND CONCLUSION

Slimming body syndrome with intellectual decline is a genetic disorder of the nervous system that is associated with decreased intellectual function, a group of known Parkinson's motor problems, visual hallucinations and sudden changes in behavior and sleep disorders. Slimming body syndrome is caused by a mental decline due to the mutation of SNCA genes that is positioned in the long arm of chromosome 4 as 4q22.1 and the SNCB gene, which is based on the long arm of chromosome number 5, is 5q35.2. There is no definitive treatment for this syndrome and all clinical measures are needed to reduce the suffering of the sufferers.

REFERENCES

1. Ferman TJ, Boeve BF, Smith GE, Lin SC, Silber MH, et al. (2011) Inclusion of RBD improves the diagnostic classification of dementia with Lewy bodies. *Neurology* 77: 875-882.
2. Hogan DB, Fiest KM, Roberts JI, Maxwell CJ, Dykeman J, et al. (2016) The prevalence and incidence of dementia with Lewy bodies: A systematic review. *Can J Neurol Sci* 43: S83-95.
3. McKeith IG, Boeve BF, Dickson DW, Halliday G, Taylor JP, et al. (2017) Diagnosis and management of dementia with Lewy bodies: Fourth consensus report of the DLB Consortium. *Neurology* 89: 88-100.
4. Morra LF, Donovick PJ (2014) Clinical presentation and differential diagnosis of dementia with Lewy bodies: A review. *Int J Geriatr Psychiatry* 29: 569-576.
5. Nalls MA, Duran R, Lopez G, Kurzawa-Akanbi M, McKeith IG, et al. (2013) A multicenter study of glucocerebrosidase mutations in dementia with Lewy bodies. *JAMA Neurol* 70: 727-735.
6. Pietrzak M, Papp A, Curtis A, Handelman SK, Katakami M, et al. (2016) Gene expression profiling of brain samples from patients with Lewy body dementia. *Biochem Biophys Res Commun* 479: 875-880.