The Relationships between Gait Impairments and Activity Limitations in People with Depressive and Related Disorders Include: Depressive Pseudodementia, Hypochondriasis, Factitious Disorder, Cognitive Dysfunction and Normal Pressure Hydrocephalus

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Abstract

According to Katzenschlager and Pirker walking speed is a sensitive indicator of general health status and it associated with life expectancy in older adults [1]. Accurate pathology diagnosis is the most important among patients exhibiting neurological disorders of gait. Several studies have investigated the relationships between gait dysfunction and participation restrictions in people with various neurological disorders. Studies show that depression has been associated with increased risk of gait impairments. This study reviewed existing evidence on gait dysfunction in neurological disease, including Depressive, Depressive Pseudodementia, Hypochondriasis, Factitious disorder, Cognitive dysfunction and Normal Pressure Hydrocephalus. The aim of this research study is to review the various neurological factors particularly relevant to depression diseases, affecting gait impairments.

Keywords: Gait Impairments; Depressive Pseudodementia; Hypochondriasis; Factitious Disorder; Cognitive Dysfunction and Normal Pressure Hydrocephalus.

Introduction

According to Pirker and Katzenschlager ‘the causes of gait disorders include neurological conditions (e.g. sensory or motor impairments), orthopedic problems (e.g. osteoarthritis and skeletal deformities) and medical conditions (e.g. heart failure, respiratory insufficiency, peripheral arterial occlusive disease and obesity)’(Pirker, et al.) [1]. Meanwhile, ‘neurological gait disorders are a common cause of falls and mortality, particularly amongst the elderly’ (Kaski and Adolfo, et al.) [2]. This study reviews the neurological aspects of gait impairments (Tan, et al & Moon, et al.), with emphasis on Depressive (Brandler, et al. & Laboni and Flint, et al.), Depressive Pseudodementia (Brenner, et al.), Hypochondriasis, (Billier, et al.) Factitious disorder (Billier, et al.), Cognitive dysfunction and Normal Pressure Hydrocephalus [3,4,5,6,7,9].

Depressive

In 2013 Brandler and et al.’ conducted a cross sectional study of the relationship between depressive symptoms and gait function, in an ambulatory community residing sample of 610 older adults (age 70 and older) who were free of dementia and MDD (Major depressive disorder) (Brandler, et al.) [5]. They made conclusion that ‘increasing depressive symptoms in community residing older adults are associated with quantitatively gait dysfunction even in the absence of major depression or dementia’ (Brandler, et al.) [5]. Simultaneously, Lord and et al. have found that ‘very mild depressive symptoms are associated with gait disturbance in early Parkinson’s disease’ (Lord, et al.) [9]. In other study (Hausdorff, et al.) have made conclusion that ‘patients with MDD and patients with bipolar disorder display gait unsteadiness’ (Hausdorff, et al.) [10].

Genetic factors

‘Recent studies have demonstrated impaired balance performance in patients with major depressive disorder (MDD)’(Deschamp, et al.) [11]. Multiple ‘genetic factors play important roles in the development of MDD’ (Major depressive disorder) according to Lohoff (Lohoff, et al.). SHTT, SHTTPLR (Brown & Harris, et al.) and SLC6A4 genes associate with MDD across numerous studies (e.g. Brown, et al., Luddington, et al., Kuzelova, et al.) [12,13,14].

Depressive Pseudodementia

According to Kennedy: Depressive Pseudodementia is a term commonly used to describe a condition whereby a
patient experiences a cognitive deficit secondary to a primary mood disorder. (Kennedy, et al.) [15]. ‘Cognitive deficits in Pseudodementia are characterized by poor effort and difficulties with attention and working memory’ (Flemming and Jr; et al.) [16]. In other word ‘Pseudeementia is a situation where a person who has depression also has cognitive impairment that looks like dementia’ ((Steckl, C; et al), Reversible Cognitive Disorder Pseudodementia) [17]. However, there are many studies showing that gait impairments coexist with depressive pseudodementia.

Genetic factors

‘Long repeat sequences in the C9ORF72 gene have cropped up in cases of multiple system atrophy (MSA) and depressive pseudodementia’ (ALZFORUM, et al.), C9ORF72 Repeats Expand into New DisordersCause, or Coincidence?) according to Bieniek, et al. [18].

Hypochondriasis

According to WilhelmSEN Hypochondriasis (Illness anxiety disorder and Somatic symptom disorder) ‘describes a persistent preoccupation with the possibility of having one or more serious and progressive physical disorders’ (WilhelmSEN et al.), and ‘defined as a chronic condition distinct from anxiety and depressive disorders’ (Simon, et al.) [19,20]. Hypochondriasis is distinguishable clinical condition (Hiller, et al) however, according to (Weck, et al) [21,22], ‘previous experiences with illness and traumatic childhood experiences did not prove to be specific risk factors for the development of hypochondriahsion’. Hypochondriasis can be accompanied by Major Depressive Disorder (MDD) (Kaphammer,et al.), According to Diagnostic and Statistical Manual of Mental Disorders (DSM5) ‘hypochondriahsion and several related conditions have been replaced by two new, empirically derived concepts: somatic symptom disorder and illness anxiety disorder’ (Mayo Clinic.) [23]. There is a strong correlation between the number of somatic symptoms and the likelihood of a depression or anxiety diagnosis (Croicu, et al.).

Genetic factors

In 2010 Holliday and et al found that: HTR2A, SERPINA6 and TPH2 are associated with somatic symptoms score [24].

Factitious disorder

According to (Uzuner, et al) ‘factitious disorder is characterized by deliberate production or imitation of physical or psychological symptoms in order to adopt the sick role’ [25]. ‘The etiology of factitious disorder is unclear’ (Osterman, et al.), however, ‘in the sense that the physical symptoms are prominently contributed by psychological factors, all somatoform disorders may be considered to be a subset of psychological factors affecting a physical condition’ (Leight, et al.) [26,27]. According to Guzman and Correll ‘patients with factitious disorder and comorbid depression have shown improvement with antidepressant therapy in addition to psychotherapy’ [28].

Genetic factors

Some studies report genetic estimates in factitious disorder.

According to (Foulds, et al) ‘Adult Onset Leukoencephalopathy with Axonal Spheroids and Pigmented Glia (ALSP) (Adams, et al.)’ [34]. This disease is recognized as presenting as a supposedly genetically confirmed hereditary disease manifesting with abnomral movements’ [30].

Cognitive Dysfunction

Cognitive dysfunction refer to deficits in attention and motor perception, learning disability, shortterm and working memory and processing speed, problem solving functions, visual or auditory processing deficits (Lam, et al.) [31]. According to Stout and Paulsen ‘changes to the motor system resulting from central nervous system damage or disease are virtually always accompanied by cognitive dysfunction’ [32]. There are a variety of different types of cognitive dysfunction e.g. ALPS or Adultonset leukoencephalopathy with axonal spheroids and pigmented glia, cognitive decline, mild cognitive impairment (Bahureksa, et al.), and Alzheimer’s disease dementia (Dorfman, et al.) [33]. Cognitive dysfunction is a common feature of Adultonset leukoencephalopathy with axonal spheroids and pigmented glia (ALSPE) (Adams, et al.) [34]. This disease is recognized as both a cognitive and a movement disorder (Ikeuchi and et al 2017). Other feature of cognitive dysfunction is Mild Cognitive Impairment (MCI). In 2015 Callisaya and et al conducted a study to determine the relationships between cognitive decline and gait slowing. They made conclusion that ‘decline in nonamnestic function (specifically executive function) was associated with decline in gait speed irrespective of the presence of baseline cognitive impairment’ (Callisaya and et al 2015) [35]. In other study in 2017 Bahureksa and et al made a conclusion that existing studies ‘provide evidence that Mild Cognitive Impairment (MCI) affects specific gait parameters’ (Bahureksa, et al) [36]. There are evidences of a direct cause relationship between the mild cognitive impairment and the development of Alzheimer’s disease (Roed, et al.).

Genetic factors

According to (Foulds, et al) ‘Adult Onset Leukoencephalopathy with Axonal Spheroids and Pigmented Gla (is) caused by a novel R782G mutation in CSF1R’ [37].

Normal pressure hydrocephalus (NPH)

According to Shprecher and et al (2008) ‘normal pressure hydrocephalus (NPH) is a syndrome of gait dysfunction and enlarged cerebral ventricles in the absence of another cause’ [38]. The characteristic psychiatric symptoms of normal pressure hydrocephalus are dementia and depression (Chopra, et al.) [39]. In 2016 (Israelsson, et al.) claimed that ‘In many dementias, depression is overrepresented, but the prevalence of depression in shunted patients with idiopathic normal pressure hydrocephalus is unknown’ [40].

Genetic factors

There are increasing evidence that iNPH may have a genetic
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Results and Conclusion

There are convincing evidences about the relationship between gait impairments and Depressive, Depressive Pseudodementia, Hypochondriasis, Factitious disorder, Cognitive dysfunction and Normal Pressure Hydrocephalus improving.

Discussion

This study was reviewed literatures including biological and psychological aspect. However, Kinesiography is important functional gait imaging modalities for study the limb mobility and neural activities. Kinesiography is the interpretation of limb moment into mathematical form. Combining Kinesiographical information with biological and psychological aspect of patient holds promise to produce and improve clinical facility. In this manner we can apply the image matching methods for the evaluation of gait impairments in patients with Depressive and related disorders.

Methods and Materials

The narrative reviews of the literatures were conducted with combination of relevant keywords. We searched the IEEE, ScienceDirect, Springer, OMICS, PubMed, PubMed Health and FINNA databases.


component (Korhonen, et al.). In 2016 (Sato, et al.) demonstrated that a ‘copy number loss in intron 2 of the SFMBT1 gene may be a genetic risk for shunt unresponsive definite iNPH’ [41-45].


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