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Frontal lobe syndrome: A mini review

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Abstract

The term "frontal lobe syndrome" often refers to a clinical illness that develops as a consequence of injury to, and dysfunction in, the prefrontal cortex. The prefrontal cortex is a huge association region that is located inside the frontal lobe. The anterior cingulate, the lateral prefrontal cortex, the orbitofrontal cortex, and the frontal poles might be implicated in this issue. Damage to higher functioning functions in the brain, such as motivation, planning, social conduct, and language or speech production, are referred to together as frontal lobe syndrome. This syndrome is a wide phrase used to characterize the damage. In this activity, the etiology, pathophysiology, and presentation of frontal lobe syndrome are discussed. Additionally, the importance of the interprofessional team in the care of this condition is highlighted.

Keywords: Frontal lobe syndrome, prefrontal cortex, prefrontal systems.

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Introduction

The frontal lobe, which is situated in front of the central sulcus, is the largest lobe of the brain in terms of neuroanatomy. It is divided anatomically and functionally into three main sections. The premotor cortex, the primary motor cortex, and the prefrontal cortex are these. Weakness and impairment in the execution of motor tasks on the contralateral side result from damage to the primary motor, additional motor, and premotor areas. A large junction of the frontal lobe known as the prefrontal cortex, which is damaged and dysfunctional, is the cause of the clinical syndrome known as frontal lobe syndrome. Anterior cingulate, lateral prefrontal cortex, orbitofrontal cortex, and anterior poles are possible related regions (Figure 1) (1).

Damage to the brain's higher functioning functions, such as motivation, planning, social behavior, and language and speech production, is referred to as frontal lobe syndrome. Frontal lobe syndrome, regardless of the cause—which can range from trauma to neurodegenerative disease—creates a challenging and complicated situation for doctors (1,2). The more complex choices and interactions required for human behavior depend heavily on the frontal lobes.

But as psychiatric disorders are increasingly treated with neurosurgery and techniques like lobotomies and leukotomies, several cases have shown profound behavioral and personality changes brought on by frontal lobe damage (3). Following his studies on the well-known Phineas Gage, who underwent a dramatic change in his behavior as a result of trauma, Harlow first coined the term "frontal lobe syndrome" to describe this collection of symptoms. Consequently, a frontal lobe abnormality can significantly alter processing, personality, and behavior that is directed toward a goal (2).

Lesions in the ventromedial orbitofrontal cortex frequently result in dramatic behavioral changes that result in impulsivity, lack of judgment, and a "frontal lobe personality" (3,4).

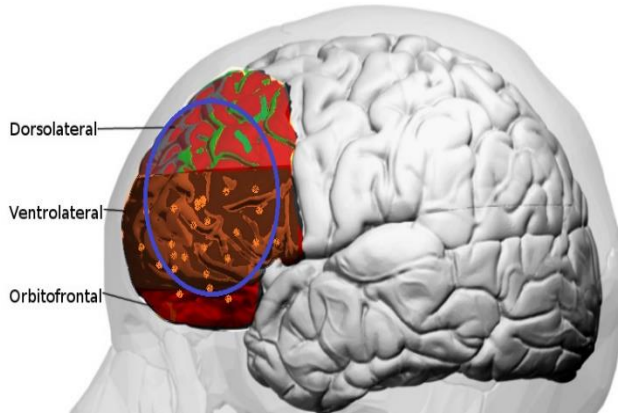

Figure 1: Frontal lobe

Deficits in working memory, rule learning, planning, attention, and motivation may result from anterior cingulate and dorsolateral lesions (5). The dorsolateral prefrontal cortex (DLPFC), in particular for monitoring and controlling the working memory content, has been shown to be essential for working memory function in recent studies. As evidenced by several cases where patients complained of attention deficit following brain trauma, the DLPFC may also have an impact on attention (6). Previous studies have looked into how DLPFC lesions may result in the "pseudo-depressive" syndrome linked to the DLPFC that is characterized by loss of initiative, decreased motivation, decreased verbal output, and behavioral sluggishness (abulia) (7). Rule learning, task switching, planning/problem solving, novelty perception, and external attention are some other processing issues (5,8). The anterior cingulate cortex is crucial for the motivation of attention, but it may also be involved in a number of psychiatric conditions like depression, PTSD, and obsessive-compulsive disorder (OCD) (9).

Etiology ve epidemiology

Numerous illnesses, such as closed head trauma (which can harm the orbitofrontal cortex), cerebrovascular disease, tumors enlarging the frontal lobe, and neurodegenerative disease, can lead to frontal lobe disorders. Other causes include HIV, multiple sclerosis, HIV, frontal lobe epilepsy, and early-onset dementia (8-10).

The underlying cause affects the frontal lobe syndrome epidemiology. Recent studies, however, contend that the prevalence of frontal lobe syndrome's neurodegenerative causes may be underestimated (10).

Pathophysiology

Lesions involving the ventromedial orbitofrontal cortex and dorsolateral convexity were prioritized based on studies in both animals and humans with frontal lobe lesions (10,11). Both lesions have different behavioral effects. Dorsolateral lesions, as was previously mentioned, can result in apathetic behavior. Ventromedial orbitofrontal lesions, on the other hand, result in impulsive, uncontrolled behaviors and potentially risky decisions. Bilateral lesions typically result in more serious deficits. Additionally, the difference between left and right frontal lesions is noted. The lesions on the left are linked to symptoms of depression, while the lesions on the right are linked to mania (11).

Histopathology

In most cases, frontal lobe syndrome brought on by neurodegenerative illnesses is brought on by one of two primary histopathologies: either alpha-synuclein or tau protein (10,11). Frontotemporal dementia, chronic traumatic encephalopathy, supranuclear palsy, corticobasal degeneration (CBD), and severe Alzheimer's disease are all examples of tauopathies that may damage the frontal lobes. When tau is hyperphosphorylated, the protein is forced to disassociate from microtubules, which results in the formation of insoluble clumps. The degree to which

neurofibrillary tangles are present is categorized according to the Braak stages. On the other hand, alpha-synuclein has been linked to Parkinson's disease, multiple system atrophy, and diffuse Lewy body dementia (7,8,10,11).

Diagnosis and evaluation

It is crucial to rule out other potential reasons of cognitive impairment before diagnosing frontal lobe syndrome. Some of these potential causes include low levels of vitamin B12, abnormal thyroid function, and syphilis serology, if available. In terms of imaging, a magnetic resonance imaging (MRI) exam could be performed to check for atrophy, hematomas, vascular and microvascular disease (12,13). In order to exclude acute bleeding or hydrocephalus, a CT scan may be performed, despite the fact that it is neither as specific nor as sensitive. Neurologists may consider a deoxyglucose PET scan if there is clinical suspicion of frontotemporal dementia (FTD). This kind of scan shows reduced activity in the frontal lobe while protecting the temporal-parietal lobes (12-14).

Differantial diagnosis

Differentials include etiologies that would also affect the frontal lobe such as (9,10,13):

. Head trauma
. Cerebrovascular accident
. Cerebral malignancies
. Frontal lobe seizures
. Hydrocephalus
. Binswanger’s encephalopathy
. Anoxic injury
. Alzheimer's disease
. Schizophrenia
. Parkinson's disease
. Huntington disease

Treatment

The type of pathology present determines the course of treatment. Acetylcholinesterase inhibitors, such as Lewy Body dementia, have shown to be effective (12). Physical and occupational therapy are examples of non-medical treatments, particularly for conditions like frontotemporal dementia. Aphasia, apraxia, and dysarthria symptoms can all benefit from speech therapy. A novel therapy for synuclein-related psychosis has been proposed: pimavanserin, a selective serotonin 5-HT2A inverse agonist (12-14). Additionally, general support, like a home caregiver, is essential as executive functions deteriorate.

Conclusions

Frontal lobe syndrome can be confused with other diseases. It is crucial to rule out other potential reasons of cognitive impairment before diagnosing frontal lobe syndrome.

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References

1. Barrash J, Stuss DT, Aksan N, Anderson SW, Jones RD, Manzel K, et al. "Frontal lobe syndrome"? Subtypes of acquired personality disturbances in patients with focal brain damage. Cortex. 2018;106:65-80.
2. Bonelli RM, Cummings JL. Frontal-subcortical circuitry and behavior. Dialogues Clin Neurosci. 2007;9:141-51.
3. Tranel D. "Acquired sociopathy": the development of sociopathic behavior following focal brain damage. Prog Exp Pers Psychopathol Res. 1994:285-311.
4. Damasio H, Grabowski T, Frank R, Galaburda AM, Damasio AR. The return of Phineas Gage: clues about the brain from the skull of a famous patient. Science. 1994;264(5162):1102-5.
5. Szczepanski SM, Knight RT. Insights into human behavior from lesions to the prefrontal cortex. Neuron. 2014;83(5):1002-18.
6. Daffner KR, Mesulam MM, Holcomb PJ, Calvo V, Acar D, Chabrerie A, et al. Disruption of attention to novel events

after frontal lobe injury in humans. J Neurol Neurosurg Psychiatry. 2000;68(1):18-24.
7. Barrash J, Bruss J, Anderson SW, Kuceyeski A, Manzel K, Tranel D, et al. Lesions in different prefrontal sectors are associated with different types of acquired personality disturbances. Cortex. 2022;147:169-84.
8. Yücel M, Wood SJ, Fornito A, Riffkin J, Velakoulis D, Pantelis C. Anterior cingulate dysfunction: implications for psychiatric disorders? J Psychiatry Neurosci. 2003;28(5):350-4.
9. Wada-Isoe K, Ito S, Adachi T, Yamawaki M, Nakashita S, Kusumi M, et al. Epidemiological survey of frontotemporal lobar degeneration in tottori prefecture, Japan. Dement Geriatr Cogn Dis Extra. 2012;2(1):381-6.
10. Guo T, Noble W, Hanger DP. Roles of tau protein in health and disease. Acta Neuropathol. 2017;133(5):665-704.
11. Kerchner GA, Tartaglia MC, Boxer A. Abhorring the vacuum: use of Alzheimer's disease medications in frontotemporal dementia. Expert Rev Neurother. 2011;11(5):709-17.
12. Cummings J, Isaacson S, Mills R, Williams H, Chi-Burris K, Corbett A, et al. Pimavanserin for patients with Parkinson's disease psychosis: a randomised, placebo-controlled phase 3 trial. Lancet. 2014;383(9916):533-40.
13. Fonseca LM, Yokomizo JE, Bottino CM, Fuentes D. Frontal lobe degeneration in adults with down syndrome and alzheimer's disease: A Review. Dement Geriatr Cogn Disord. 2016;41(34):123-36.
14. Mumoli N, Pulerà F, Vitale J, Camaiti A. Frontal lobe syndrome caused by a giant meningioma presenting as depression and bipolar disorder. Singapore Med J. 2013;54(8):e158-9.