Journal of Dementia

Gabriela, J Dement 2023, 7:4

Editorial Open Access

A Breif Note on Behavioral Variant Frontotemporal Dementia and Basic Psychiatric Symptoms Have Neuroanatomical Components

Filip Gabriela*

Melbourne Neuropsychiatry Centre, University of Melbourne, Australia

Abstract

Although primary psychiatric disorders (PPDs) and behavioral variant frontotemporal dementia (bvFTD) share many of their symptoms, their structural anatomical changes have not been thoroughly studied. We included studies on bvFTD, schizophrenia, bipolar disorder, and autism spectrum disorder that 1) reported the coordinates of the regional gray matter volumes (GMVs) and 2) used voxel-based morphometry analysis to assess regional GMVs in this magnetic resonance imaging-based meta-analysis. Between patients and control subjects, clusters of coordinate-based changes in the GMVs were compared in separate analyses (n = 24,183), and overlapping brain regions between each PPD and bvFTD were examined. The transdiagnostic brain alterations in bvFTD and PPD consist of GMV alterations in the temporal lobe, amygdala, and insula, as well as the prefrontal and anterior cingulate cortices. Our cross-disorder approach would provide new insights into the relationship between bvFTD and PPD, and our meta-analysis revealed significant anatomic overlap that paves the way for future investigations of shared pathophysiological pathways.

Keywords: Psychiatric disorders; Schizophrenia; Bipolar disorder; Metabolic diseases

Introduction

Neurodegenerative disease known as frontotemporal dementia (FTD) primarily affects the frontal and/or temporal lobes The behavioral variant (bvFTD), which manifests as disinhibition, social awkwardness, loss of insight, apathy, loss of empathy, stereotypical behavior, and changes in eating habits, is the most prevalent subtype [1]. A gradual loss of social cognition, which in turn affects aspects of behavior and personality, is one of the earliest and most important symptoms of bvFTD. Several major primary psychiatric disorders (PPDs), including schizophrenia (SZ), bipolar disorder (BD), and autism spectrum disorder (ASD), share a striking clinical similarity with bvFTD. More specifically, one of the main characteristics of PPD is impaired social cognition (6). As a result, bvFTD and major PPDs may both be categorized as social brain disorders [2]. In addition, mania's euphoric state and lack of insight can strongly resemble bvFTD in daily clinical practice. At long last, both the positive and negative side effects of SZ (e.g., fancies and pipedreams versus social withdrawal, lack of unconstrained discourse, and solidness, separately) are basically the same as what is seen in bvFTD. Due to similar and overlapping diagnostic criteria for bvFTD and various PPDs, approximately 50% of patients with bvFTD receive a prior psychiatric diagnosis. Carriers of a C9orf72 repeat expansion show a stronger correlation between psychiatric symptoms and neurodegenerative disease [3]. SZ and BD are more common in family members of C9orf72 mutation carriers, but C9orf72-related FTD can present with SZ, BD, or ASD symptoms (11–15). In addition, FTD neuropathology may be underlying in young cases with SZ and BD diagnoses. Independent authors have hypothesized a shared neurobiological background between bvFTD, SZ, BD, and ASD on the basis of this empirical overlap. However, they have not yet tested their hypotheses [4].

Method

In view of the clinical cross-over and given the huge primary changes in the frontotemporal cerebrum areas in patients with a PPD in enormous scope studies yielded by the Puzzler (Improving Neuro Imaging Hereditary qualities through Meta Examination) Consortium, in this cross-jumble investigation, we conjecture that bvFTD and PPDs share an organic weakness of explicit neuroanatomical organizations [5]. It is important to find common neuroanatomical vulnerabilities between bvFTD and PPDs because this could help build a conceptual framework for understanding how these disorders are related to one another and whether they share pathophysiological pathways that could be targeted by treatment [6]. The neuroimaging technique known as voxel-based morphometry (VBM) is used to measure the structure of gray matter (GM). Using a voxelwise, coordinate-based meta-analytic method, we sought to identify the overlapping GM differences between bvFTD and PPD, including SZ, BD, and ASD, in this cross-disorder comparison [7].

The Preferred Reporting Items for Systematic Reviews and MetaAnalyses (PRISMA) statement was followed during the execution of this meta-analysis. The Medline, Embase, and BrainMap databases were used to collect the studies for this meta-analysis, which covered the literature up until April 2020. Our meta-analysis was registered on the Open Science Framework [8]. We didn't combine any of the analyses because we knew that different approaches could have an impact on the volumetric results. As a result, we concentrated on whole-brain VBM analysis, which is widely utilized in the neuroimaging scientific community to measure GM volume (GMV). As a result, when measuring cortical thickness with FreeSurfer, we excluded GM modifications to prevent any misinterpretation [9].

Result

Studies needed to meet the following inclusion criteria before they could be included in our meta-analysis: 1) compared healthy control subjects to patients using structural neuroimaging; 2) utilized the

***Corresponding author:** Filip Gabriela, Melbourne Neuropsychiatry Centre, University of Melbourne, Australia, E-mail: filip4@gmail.com

Received: 21-June-2023, Manuscript No: dementia-23-103913; **Editor assigned:** 24-June-2023; Pre QC No: dementia-23-103913 (PQ), **Reviewed:** 8-Jul-2023; QC No: dementia-23-103913; **Revised:** 13-Jul-2023; Manuscript No: dementia-23-103913 (R) **Published:** 18-Jul-2023, DOI: 10.4172/dementia.1000165

Citation: Gabriela F (2023) A Breif Note on Behavioral Variant Frontotemporal Dementia and Basic Psychiatric Symptoms Have Neuroanatomical Components. J Dement 7: 165.

Copyright: © 2023 Gabriela F. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Rascovsky, Neary, and McKhann, bvFTD diagnostic criteria; used the revised ASD Autism Diagnostic Interview; or for SZ, BD, and ASD, used the DSM-III, DSM-IV, DSM5 (10), or ICD-10; 3) carried out a GMV VBM analysis; 4) reported coordinates in Talairach stereotactic standard space or the Montreal Neurological Institute (MNI) 5) Only patients over the age of 16 were included; 6) Peak coordinates of statistical significance at the whole-brain level were reported by GM alterations; what's more, 7) were written in English. Studies were barred when 1) no unique information were announced, for instance, letters to the manager, meta-investigations, or survey studies, and 2) the review test covered with those of another distribution. In the event of sample duplication, the study with the largest sample size was chosen from those from the same institution or cohort at the same time [10-13].

Discussion

All citations in our search were registered using the Endnote database. Based on similarities in authorship, study description, year of publication, and journal, duplicate studies were removed. The citations' titles and abstracts were then scrutinized by CT and HU, two independent authors, to determine their suitability for inclusion. Consensus or the decision of a third author was used to settle disagreements between authors (YALP). After that, we looked at the full-text articles with the relevant citations to see if the study met the inclusion criteria.

Clusters of voxels with changes in regional GMVs were compared between the patient groups and healthy control subjects in separate analyses. Each p value and the activation likelihood estimation (ALE) thresholds in the results were reported at a cluster-forming threshold; bunches with more noteworthy Lager values than this limit were thought of as genuinely critical) registered utilizing a p , .05, misleading disclosure rate adjusted (without any presumptions to connections inside the dataset), and a moderate least bunch volume of 200 mm3 utilizing BrainMap's GingerALE Pinnacle facilitates for GMV were separated from qualified examinations and were switched over completely to MNI152 format utilizing the Lancaster change before investigation (24). GingerALE 3.0.2 of BrainMap was used to enter the MNI coordinates for the data.

Conclusion

The procedure's specifics are available on the website at http://brainmap.org/ale/index.html. In brief, the most recent ALE algorithm in GingerALE was used to perform meta-analysis calculations. The coordinates reported by the studies included in this meta-analysis were used to estimate the likelihood of anatomical differences between groups. Focuses at each voxel were combined to create a modeled map. A cluster-level, family wise error–corrected p value of.05. was used to threshold the statistical maps. The direction of the gauged focus was produced for each bunch. The MNI location of the cluster in the MNI152 atlas was then assigned to the maximum ALE value and its coordinates within the cluster. GingerALE created unique datasets for each diagnostic group using the collected coordinates. In order to investigate GM alterations within each disorder group, separate single-dataset analyses were carried out. Later breaking down the single dataset for each analytic gathering, we performed 3 pairwise combination examinations to concentrate on the cross-over between 1) bvFTD and SZ, 2) bvFTD and BD, and 3) bvFTD also, ASD. We used a voxel threshold of p.05 and a cluster-forming threshold of p.001 for the conjunction analyses.

The Joanna Briggs Institute quality assessment tool was used to evaluate the study quality of each and every article that was included. The rigor of the inclusion criteria, subject selection, measurement of exposure, condition, identification of confounders, strategies for confounders, measurement of outcome, and statistical analysis are all evaluated using the nine-point checklist. The measurement of exposure has not been taken into account in the final quality assessment because of the methodology of our included studies. In VBM studies, the Joanna Briggs Institute quality assessment tool is frequently used as a recommended methodological quality (risk of bias) assessment tool.

References

1. Chambers CD, Bellgrove MA, Stokes MG, Henderson TR, Garavan H, et al. (2006) Executive "brake failure" following deactivation of human frontal lobe. J Cogn Neurosci 18: 444-455.
2. Chun J, Karam ZN, Marzinzik F, Kamali M, O'Donnell L, et al. (2013) Can P300 distinguish among schizophrenia, schizoaffective and bipolar I disorders? An ERP study of response inhibition. Schizophr Res 151: 175-184.
3. Krauss JK, Regel JP, Droste DW (1997) Movement disorders in adult hydrocephalus. Mov Disord 12: 53-60.
4. Tarkowski E, Tullberg M, Fredman P (2003) Normal pressure hydrocephalus triggers intrathecal production of TNF-alpha. Neurobiol Aging 24: 707-714.
5. Urzi F, Pokorny B, Buzan E (2020) Pilot Study on Genetic Associations With Age-Related Sarcopenia. Front Genet 11:615238.
6. Starkweather AR, Witek-Janusek L, Nockels RP, Peterson J, Mathews HL (2008)The Multiple Benefits of Minimally Invasive Spinal Surgery: Results Comparing Transforaminal Lumbar Interbody Fusion and Posterior Lumbar Fusion. J Neurosci Nurs 40:32–39.
7. Bauer JM, Verlaan S, Bautmans I, Brandt K, Donini LM, et al. (2015) Effects of a vitamin D and leucine-enriched whey protein nutritional supplement on measures of sarcopenia in older adults, the PROVIDE study: a randomized, double-blind, placebo-controlled trial. J Am Med Dir Assoc 16:740–747.
8. Inose H, Yamada T, Hirai T, Yoshii T, Abe Y, et al.(2018) The impact of sarcopenia on the results of lumbar spinal surgery. Osteoporosis and Sarcopenia 4:33–36.
9. DigheDeo D, Shah A (1998) Electroconvulsive Therapy in Patients with Long Bone Fractures. J ECT 14: 115–119.
10. Takahashi S, Mizukami K, Yasuno F, Asada T (2009) Depression associated with dementia with Lewy bodies (DLB) and the effect of somatotherapy. Psychogeriatrics 9: 56–61.
11. Bellgrove MA, Chambers CD, Vance A, Hall N, Karamitsios M, et al. (2006) Lateralized deficit of response inhibition in early-onset schizophrenia. Psychol Med 36: 495-505.
12. Carter CS, Barch DM (2007) Cognitive neuroscience-based approaches to measuring and improving treatment effects on cognition in schizophrenia: the CNTRICS initiative. Schizophr Bull 33: 1131-1137.
13. Gupta S, Fenves AZ, Hootkins R (2016) The Role of RRT in Hyperammonemic Patients. Clin J Am Soc Nephrol 11:1872-1878.